

IDENTIFYING MECHANISMS THAT PREDICT LOSS OF CONTROL (LOC)  
EATING USING ECOLOGICAL MOMENTARY ASSESSMENT

by

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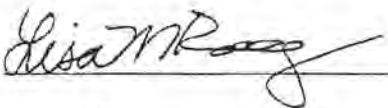
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## **ABSTRACT**

Title of Dissertation: Identifying Mechanisms that Predict Loss of Control (LOC) Eating  
Using Ecological Momentary Assessment

Lisa M. Ranzenhofer, Doctor of Philosophy, 2013

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Binge eating disorder (BED) is the most prevalent eating disorder in the U.S., with estimates ranging from 2.5% in the community to 30% in overweight, treatment-seeking individuals. In youth, full-syndrome BED is rare, however rates of binge eating (BE) and loss of control (LOC) eating in absence of the full syndrome range from 6 - 40%, with the highest rates observed in overweight adolescent girls. Given ambiguity surrounding what amount of food constitutes a 'binge' among growing youth of varying ages and energy requirements, the experience of LOC is considered the most salient feature of pathological eating, rather than the amount of food consumed. In youth, LOC eating is associated with elevated eating pathology and general psychological symptoms and is predictive of excess weight gain and worsening eating pathology. Despite its adverse correlates, little is known about the etiology of LOC eating, specifically, the moment-to-moment processes promoting LOC episodes. One model used to explain LOC eating is the interpersonal model, positing that interpersonal problems precede and

predict negative affect, which in turn precedes and predicts episodes of LOC eating. Despite support for links between model components, no study has directly tested the interpersonal model in a temporally sensitive and ecologically-valid manner. The present study examined the feasibility of using ecological momentary assessment to examine the interpersonal model of LOC among overweight adolescent girls who experience LOC eating. Additionally, because youth and individuals with eating pathology are thought to have difficulty identifying and describing their emotional states, negative affect was also assessed using a physiologic proxy, heart rate variability, as an index of emotional stress. Thirty participants who experienced LOC completed ecological momentary assessments multiple times per day for a period of two weeks. Heart rate variability was assessed on a subset of two days of the total monitoring period. Results suggested that interpersonal problems were the strongest predictor of LOC eating. Negative affect was marginally predictive of LOC eating, although it was not found to mediate the relationship between interpersonal problems and LOC. Physiologic data revealed elevated heart rate and lower heart rate variability at times of high self-reported interpersonal problems. Furthermore, trajectories of heart rate and heart rate variability prior to LOC episodes differed significantly from those preceding non-LOC episodes, and were similar to autonomic characteristics of a stress response. Although the full interpersonal model was not supported, links between components of the interpersonal model revealed that interpersonal and physiologic factors appear to contribute to the momentary etiology of LOC episodes. Further analyses within an adequately powered study are needed before definitive conclusions can be drawn.

## TABLE OF CONTENTS

LIST OF TABLES .....	9
LIST OF FIGURES .....	10
CHAPTER 1: BACKGROUND.....	12
Binge Eating Disorder.....	12
Demographics .....	13
Prevalence.....	13
Race .....	13
Association with obesity.....	14
Course.....	14
Health-related and psychological correlates .....	15
Eating pathology.....	15
Psychiatric comorbidity .....	16
Impairment and quality of life .....	17
Binge Eating in Youth.....	18
Demographics .....	18
Prevalence.....	18
Race/ethnicity.....	18
Association with obesity.....	19
Course.....	19
Health-related and psychological correlates .....	19
Loss of Control Eating .....	20
Assessment of eating episodes in youth.....	20
Qualitative features of LOC eating in youth.....	21
Health-related and psychological correlates .....	22
Interpersonal model of LOC .....	23
Interpersonal problems and negative affect .....	24
Theoretical underpinnings.....	24
Evolutionary theories.....	24
Interpersonal theory.....	25
Adolescents' interpersonal contexts .....	27
Empirical Support.....	28
Peer relationships .....	28
Parental relationships.....	30
Salience of interpersonal relationships among overweight adolescents .....	31
Negative affect and LOC.....	32
Theoretical underpinnings.....	32
Empirical underpinnings.....	33
Self-report studies .....	33
Laboratory studies.....	34
Ecological momentary assessment studies .....	35
Full interpersonal model.....	36

Empirical support.....	36
Measurement of Affect.....	37
Alexithymia .....	37
Utility of physiological index of affect.....	38
Human Emotions and Physiology .....	38
Hypothalamic-pituitary-adrenal axis .....	38
Structure and function .....	38
HPA axis and the interpersonal model.....	40
Autonomic nervous system .....	42
Structure and function .....	42
Clinical relevance.....	45
ANS and the interpersonal model.....	47
Physiological biomarkers as indices of emotional responding .....	50
Ecological Momentary Assessment.....	51
Momentary nature of interpersonal model .....	51
Self-report measures .....	52
Physiologic measures .....	52
CHAPTER 2: RATIONALE AND SPECIFIC AIMS.....	55
Rationale.....	55
Specific Aims.....	55
General Analytic Strategy.....	56
Hypotheses and Statistical Analyses .....	58
Aim 1.....	58
Hypothesis 1. ....	58
Hypothesis 1a. ....	58
Hypothesis 1b.....	59
Hypothesis 1c.....	59
Hypothesis 1d.....	59
Aim 2.....	60
Hypothesis 2. ....	60
Aim 3.....	62
Hypothesis 3. ....	62
Hypothesis 3a. ....	63
Hypothesis 3b.....	64
Hypothesis 3c. ....	65
Hypothesis 3d.....	66
CHAPTER 3: RESEARCH DESIGN AND METHODOLOGY .....	68
Overview .....	68
Participants.....	68
Recruitment.....	70
Procedures .....	71
Part 1: Baseline screening appointment .....	72
Part 2: EMA practice period.....	72
Part 3: Data collection .....	73
Measures.....	73

Body composition.....	73
Interviews.....	74
Questionnaires.....	75
EMA.....	75
Protocol.....	76
Measures captured.....	78
Holter monitoring.....	81
Data collection.....	81
Data analysis.....	81
Evaluation.....	84
Power Analysis.....	85
CHAPTER 4: RESULTS.....	86
Demographics and Baseline Measures.....	86
Demographics.....	87
Loss of control eating.....	87
Psychological measures.....	88
Beck Depression Inventory (BDI-II).....	88
State Trait Anxiety Inventory (STAI-C).....	88
Kiddie Schedule for Affective Disorders and Schizophrenia (KSADS).....	88
Data Cleaning and Reduction.....	89
Outlier Screenings.....	89
EMA.....	90
HRV.....	91
Protocol.....	92
Hypothesis Testing.....	93
Aim 1.....	93
Hypothesis 1a.....	93
Hypothesis 1b.....	95
Hypothesis 1c.....	97
Hypothesis 1d.....	97
Aim 2.....	101
Hypothesis 2.....	101
24-hour HRV and trait negative affect.....	102
24-hour HRV and daily affect.....	105
Momentary HRV and state negative affect.....	106
Aim 3.....	110
Self-report variables.....	110
Physiologic variables.....	111
Hypothesis 3a.....	113
Self-report.....	113
Hypothesis 3b.....	114
Self-report.....	114
Physiologic.....	115
Hypothesis 3c.....	118
Self-report.....	118

Physiologic .....	119
Summary .....	122
Hypothesis 3d .....	123
CHAPTER 5: DISCUSSION .....	124
Aim one .....	124
Aim two .....	134
Aim three .....	138
Clinical implications .....	147
Strengths and limitations .....	149
Conclusions .....	155
REFERENCES .....	195



## LIST OF TABLES

Table 1.	Participant demographics .....	157
Table 2.	Reliability analyses .....	158
Table 3.	Compliance.....	160
Table 4.	Acceptability .....	161
Table 5.	Regression analyses for prediction of compliance .....	162
Table 6.	Heart rate and heart rate variability .....	163
Table 7.	Aggregate interpersonal model variables by sub-threshold BED status.....	164
Table 8.	Estimates of fixed effects from multilevel models of the relationships between momentary negative affect and heart rate, <sup>a</sup> SDNN, <sup>b</sup> RMSSD, <sup>c</sup> pNN50 and <sup>d</sup> HF/HF+LF power, examined in main effects models, split file models, and interaction models.....	165
Table 9.	Multilevel models of heart rate, <sup>a</sup> SDNN, <sup>b</sup> RMSSD, <sup>c</sup> pNN50, and <sup>d</sup> HF/HF+LF over time following high and low interpersonal problem ratings.....	172
Table 10.	Multilevel models of heart rate, <sup>a</sup> SDNN, <sup>b</sup> RMSSD, <sup>c</sup> pNN50, and <sup>d</sup> HF/HF+LF over time prior to LOC and non-LOC eating episodes .....	175

## LIST OF FIGURES

Figure 1. Interpersonal model of Loss of Control (LOC) eating, positing that interpersonal problems precede and predict negative affect, which in turn precedes and predicts LOC eating. Negative affect is thought to serve as a mediating variable between interpersonal problems and LOC eating. ....	178
Figure 2. Model of negative affect as a mediator of social problems and LOC eating, controlling for age, sex, race, pubertal status, and BMI-Z score. ....	178
Figure 3. Steps involved in testing mediation. Path c: demonstrate that the initial variable (X) is correlated with the outcome variable (Y). Path a: demonstrate that the initial variable (X) is correlated with the mediator variable (M). Path b: while controlling for the initial variable, show that the mediator variable (M) is correlated with the outcome variable (Y). Path c': to establish the degree to which M is a mediator between X and Y, calculate the degree to which the correlation between X and Y is reduced when controlling for M. ....	180
Figure 4. Depression and anxiety scores on the Beck Depression Inventory-II (BDI-II) and the State-Trait Anxiety Inventory-Child, trait version, for sub-threshold BED and non-BED groups .....	181
Figure 5. Total, internalizing, and externalizing psychiatric symptoms as measured by the Kiddie Schedule for Affective Disorders and Schizophrenia (KSADS) for sub-threshold BED and non-BED groups .....	182
Figure 6. Reasons for non-compliance reported by adolescents.....	183
Figure 7. Relationships between daily normalized HF power and total psychiatric symptoms and internalizing symptoms .....	184
Figure 8. Relationships between daily normalized HF power and dimensions of state affect including negative affect, positive affect, and boredom.....	185
Figure 9. Relationship between momentary negative affect (log transformed) and heart rate for random, before-meal, and after-meal recordings.....	186
Figure 10. Relationship between momentary positive affect and the proportion of HF power for random, before-meal, and after-meal recordings.....	187
Figure 11. Interpersonal model: path c. Relationship between interpersonal problems (X) at time 1 and LOC eating (Y) at time 2. In this analysis, interpersonal problems directly preceded LOC ratings, with measurements occurring approximately one half hour apart.....	188
Figure 12. Interpersonal model: path a: Relationship between interpersonal problems (X) at time 1 and state affect (M) at time 2, with measurements occurring approximately one half hour apart. Shown are negative affect, positive affect, and boredom. ....	189
Figure 13. Interpersonal model: path b: Relationship between negative affect (M) and LOC Eating (Y). The first graph depicts the relationship between negative affect at time 1 and LOC Eating at time 2 (n = 30, k = 426). The second graph depicts the relationship between negative affect at time 2, controlling for negative affect at time 1 (thereby representing the change in negative affect) and LOC Eating at time 3 (n = 29, k = 184). ....	190



Figure 14. Trajectories of heart rate, SDNN, RMSSD, pNN50, and HF-HRV during the 3 hours preceding after-meal ratings for “high” and “low” LOC episodes.....	191
Figure 15. Summary of findings from aim 3 hypotheses 1 – 3 for negative affect, using (a) all cases and (b) only cases included in the mediation model.....	192
Figure 16. Summary of findings from aim 3 hypotheses 1 – 3 for boredom, using (a) all cases and (b) only cases included in the mediation model. ....	193
Figure 17. Summary of findings from aim 3 hypotheses 1 – 3 for positive affect, using (a) all cases and (b) only cases included in the mediation model.....	194

## **CHAPTER 1: BACKGROUND**

### **BINGE EATING DISORDER**

Binge eating disorder (BED) entered the DSM-IV as a putative diagnosis in 1994 to capture the aberrant eating pattern characterized by recurrent episodes of BE in the absence of regular compensatory behaviors (8). Binge eating (BE) is defined as eating, in a discrete period of time, an amount of food that is definitely larger than the amount of food that most people would eat in a similar amount of time (termed “unambiguously large”), while experiencing a sense of being unable to stop eating or control what or how much one is eating (termed “loss of control” (LOC)). Eating episodes are thus classified on the basis of two criteria: episode size and the presence or absence of LOC during eating. Three types of eating episodes are often discussed in the eating disorders literature: objective bulimic/binge episodes, subjective bulimic/binge episodes, and objective overeating. Episodes that are considered unambiguously large and involve the experience of LOC are termed “objective bulimic/binge episodes.” When occurring at least twice per week over a three month period, these episodes comprise BED (9). By contrast, “subjective bulimic episodes” denote episodes in which a sense of LOC is experienced but the amount of food is not viewed as large, and “objective overeating” describes episodes in which the amount of food consumed is considered large, but in which LOC is not experienced. In order to meet criteria for BED, objective binge episodes are accompanied by at least three of the following features: eating fast, eating until uncomfortably full, eating large amounts of food when not hungry, eating alone due to embarrassment regarding how much one is eating, and feeling disgusted, depressed, or very guilty after overeating (Criteria B), as well as significant distress regarding BE

(Criteria C). Binge episodes are not followed by regular use of inappropriate compensatory behaviors such as purging or excessive exercise (Criteria E).

## **Demographics**

### ***Prevalence***

Studies throughout the past several decades suggest that BED affects between 1½-6% of the general population (83; 111; 191; 192). Although women experience higher rates of BED than men, a large number of men are also impacted, with a female to male ratio of approximately 3:2 (4; 34; 99).

### ***Race***

Despite traditional conceptions that eating disorders (e.g. anorexia nervosa (AN) and bulimia nervosa (BN)) predominantly impact upper-class Caucasian girls and women, BED appears to be equally, if not more, common across racial and ethnic minority groups. Given higher prevalence rates of obesity among ethnic minority groups (66), and the strong association between BED and obesity, higher prevalence of BED among racial and ethnic minority groups may be expected. Indeed, studies conducted among diverse population and community samples have generally revealed equal or higher prevalence rates of BED among racial and ethnic minority groups. For instance, in an early population-based survey, Bruce and Agras found similar prevalence rates of BED among African American (5%), Hispanic (5%), and Caucasian women (2%) (31). Another community survey similarly found equal prevalence of current (within three-months) BE across women of varying racial and ethnic sub-groups, although recurrent BE was more common among African American women (199). In an epidemiologic, national household survey conducted among Latinos in the U.S., current (within 12-

month) and lifetime BED prevalence rates were found to be 1.2% and 2.3%, respectively. Current and lifetime prevalence rate of BE were 3.2% and 5.8%, respectively, aligning with population estimates (99).

### *Association with obesity*

Although obesity is not a requisite criterion for BED, BED is more prevalent among overweight individuals than in the general population. Population-based studies suggest a strong correlation between BED and obesity (83; 99). Although 70% of individuals meeting criteria for BED fall into an obese category (83), most individuals who are obese do not meet criteria for BED. In a non-clinical sample, BED prevalence among obese individuals was found to be only 5% (191). Yet, prevalence estimates of BED are consistently higher among obese, treatment-seeking individuals (40), consistently falling around 30% across samples (106; 192; 241).

### *Course*

Whereas AN and BN commonly emerge during adolescence and young adulthood, individuals with BED typically present for treatment later in the life course. Across community and treatment-seeking samples comprised of adults over 18 years, the mean age of individuals with BED generally ranged between mid-30s to mid-40s (82; 83; 241). In the National Comorbidity Survey Replication, a nationally representative face-to-face household survey, Hudson and colleagues found mean ages of onset of AN, BN, and BED to be 18.9, 19.7, and 25.4 years, respectively (99). BE in absence of BED tends to precede the onset of BED, with a mean age of onset of 22.4 years (99).

In absence of intervention, the mean duration of BED as reported by individuals in the National Comorbidity Survey replication was 8.1 years (99). In contrast, studies

employing repeated assessments (rather than retrospective self-report of the duration of illness) suggest that the disorder may naturally recur and remit over time. Among a community sample of women with BED, about half continued to experience the full-syndrome when assessed 6 months later (32). In a separate sample of women aged 18 - 35 with an initial BED diagnosis, 18% continued to meet a diagnosis for any eating disorder, and 10% continued to meet diagnostic criteria for BED, at five-year follow up (62). Comprising the longest follow up of BED known to date, a recent study published 12-year BED outcomes in 68 female inpatients. Findings suggest that while only 8% of women continued to meet full criteria for BED, 36% maintained an eating disorder diagnosis of any kind (64). Finally, the placebo response rate for BED, calculated by assessing the proportion of patients who no longer met BED diagnostic status or who markedly reduced their BE (BE) frequency following treatment with a placebo, was approximately 30% (103), which is similar to the placebo response rate for major depressive disorder but higher than for BN. Participants who responded to placebo had less severe baseline pathology. Further, many placebo-responders experienced continued symptoms at one-year follow-up (103).

### **Health-related and psychological correlates**

BED is considered a disorder of high clinical relevance given its association with obesity and extensive obesity-related comorbidities (83; 185; 191; 192; 199). The clinical relevance of BED is also underscored by high rates of eating-related and general psychopathology that accompany BED.

### ***Eating pathology***

Individuals with BED are distinguishable from their overweight counterparts without BED by higher rates of eating-related pathology, including emotional eating, restraint, and weight and shape concerns (29; 53; 237). Among 168 adults participating in commercial weight loss programs, those with BED endorsed higher levels of concern regarding their body shape and weight, and greater likelihood of eating in response to emotions, compared to overweight individuals without an eating disorder (53).

Furthermore, comparisons of women with AN, BN and BED suggest that eating disordered attitudes and cognitions in BED are equally severe as among women with AN and BN (237). Using a clinical interview to assess BED status and eating disorder pathology, BED patients reported equivalent importance of shape and weight for self-evaluation, preoccupation with shape and weight, and overall shape and weight concerns, as compared to those with AN and BN (237). Taken together, data suggests that BED patients are both distinct from overweight individuals without BED on the dimension of eating pathology and demonstrate similar eating pathology as compared to those with other eating disorders. Such findings underscore the status of BED as an eating disorder.

### ***Psychiatric comorbidity***

Individuals with BED also have higher rates of co-morbid psychiatric disorders compared to obese controls, including both axis I and axis II disorders (83; 192; 199; 241). In the National Comorbidity Survey replication, 80% of patients meeting criteria for BED also met criteria for at least one additional DSM-IV-TR psychiatric diagnosis (99). In a study employing psychiatric interviews to assess BED and co-morbid psychiatric diagnoses, Yanovski and colleagues (1993) found BED to be co-morbid with major depressive disorder, panic disorder, BN, and borderline personality disorder, but



not with substance abuse or generalized anxiety (241). Furthermore, BED participants were more likely to have undergone therapy and have a family history of substance abuse. By contrast, in a study of severely obese patients undergoing gastric bypass surgery, there were no differences between BED and non-BED patients in terms of DSM-IV-TR psychiatric morbidity (98).

Across studies, major depressive disorder is the most common co-morbid diagnosis among individuals with BED, with one study suggesting that major depressive disorder is nearly three times more prevalent among individuals with BED compared to non-BED obese controls (220). Among a large ( $n = 404$ ) sample of BED patients, those with current major depressive disorder (MDD) reported greater eating disorder pathology and lower self-esteem (82) compared to patients without current MDD. By contrast, BED patients with a lifetime history of (but not current) MDD did not differ on any dimension compared to those without history of a psychiatric disorder.

### ***Impairment and quality of life***

In addition to its association with psychiatric diagnoses, individuals with BED often report poorer quality of life and greater impairments in social and work domains compared to obese individuals without BED (98; 166; 192). For instance, among a large sample ( $n = 1,785$ ) of patients from 18 weight-control clinics, patients with and without BED were compared on various dimensions of role impairment. Findings indicated that patients with BED experienced greater impairment in relationships and work as a result of being upset by issues of eating and weight compared to non-BED patients. Individuals with BED also reported greater interference of weight/shape with “feeling good” (192). Similarly, patients with extreme obesity who underwent gastric bypass surgery differed

according to BED status in terms of their role impairment in physical, emotional, and social functioning, with BED subjects reporting poorer functioning (98). Finally, in terms of quality of life, with the exception of physical functioning, BED subjects reported decrements in nearly every domain including work, public distress, sexual life, self-esteem, and total quality of life, as compared to obese subjects without BED (166).

## **BINGE EATING IN YOUTH**

### **Demographics**

#### ***Prevalence***

Rates of BED among youth are lower than those seen among adults, with estimates ranging from less than 1% among adolescents in the community (104) to around 6% among overweight adolescents seeking residential treatment for obesity (43). Although few children and adolescents meet full criteria for BED, rates of BE in absence of the full syndrome are estimated to be between 6 - 40% across samples (37; 68; 104; 147). Similar to adults, many studies among adolescents suggest that girls are more likely to engage in BE than boys (65; 148), and overweight adolescent girls report the highest rates of BE (148).

#### ***Race/ethnicity***

Binge eating is reported with similar prevalence among Caucasians and ethnic/racial minority groups. Among a large community sample of adolescent girls, French and colleagues found similar prevalence of BE across racial groups: 33.6% among Asians, 30.6% among Caucasians, 29% among American Indians, 25.2% among Latinos, and 23% among African Americans (68). By contrast, Johnson and colleagues examined prevalence of BE among both sexes, and found higher prevalence among African American boys (26%)



compared to other demographic groups (African American girls (17%), Caucasian boys (19%) and Caucasian girls (18%) (104).

### ***Association with obesity***

Similar to adults, BE is strongly correlated with weight status, with higher rates of BE seen among overweight youth (147; 148). Among overweight adolescents seeking weight loss treatment, approximately one third report BE (43; 50; 73; 102).

### ***Course***

Most adult studies cite the emergence of BED during early adulthood; however, studies among youth suggest that binge and LOC eating behavior (in absence of full-syndrome BED) emerge much earlier. Among 6 - 13 year old children queried regarding when they first experienced LOC eating, the mean age of onset was  $8.4 \pm 1.9$  years (206), suggesting that LOC eating may often begin as early as middle-childhood.

### **Health-related and psychological correlates**

Similar to adults, BE in youth is associated with a number of adverse psychosocial correlates. Adolescents who report BE are more likely to manifest a variety of problems including disordered eating cognitions, depressive symptoms, poorer family and social functioning, and emotional stress than those without BE (68; 104; 126; 193). In community samples, BE is associated with depressive symptoms across studies (104; 126). French and colleagues found BE to be associated with peer acceptance concern and emotional stress in many (Caucasian, African American, Asian), but not all (Latino, American Indians) ethnic groups (68). Among a large sample of adolescents from the community (ages 12-19), BE was associated with a number of specific eating behaviors including skipping meals, snacking, eating sweets, and having an “unbalanced diet” (126). Prospectively, BE in youth

aged 6 - 12 was associated with greater weight gain over a 4 year follow-up interval (205). Treatment-seeking adolescents with BED are thought to have significantly higher negative mood, trait anxiety, and eating disorder pathology (eating, shape, and weight concerns), compared to adolescents with sub-threshold BED or no eating pathology (73). Similarly, among overweight treatment-seeking children and adolescents, those with BE had significantly poorer global self-esteem and perceptions of physical appearance, as well as elevated eating, shape and weight concerns (43).

## **LOSS OF CONTROL EATING**

### **Assessment of eating episodes in youth**

Defining what constitutes an episode of BE during childhood and adolescence is limited by difficulty estimating what amount of food is considered ‘unambiguously large’ for growing youth of varying activity levels and energy requirements. For growing teens, differentiating between objective and subjective bulimic episodes on the basis of size is considered a difficulty in assessment of pathological eating. Thus, many researchers consider the experience of LOC, rather than the amount of food per se, as the key factor in defining pathological overeating among youth. LOC eating refers to episodes of eating characterized by an inability to control what or how much is being eaten, regardless of the amount of food reportedly consumed (204). According to this classification method for aberrant eating episodes, *objective* and *subjective* bulimic episodes are both categorized as LOC eating.

Indeed, studies suggest few differences between youth who describe LOC eating episodes characterized by the consumption of an “unambiguously large” amount of food

versus episodes characterized by consuming an amount of food not considered unambiguously large. Among adolescents ages 13 - 17 who reported either BED, subclinical BE, recurrent overeating, or no aberrant eating, those endorsing any form of LOC eating (full syndrome or subclinical) had greater weight concerns compared to either group without LOC (overeating, no aberrant eating) (77). Similarly, adolescents with full-syndrome and subclinical BE had significantly greater depressive symptoms compared to adolescents with no aberrant eating, although only the full-syndrome group differed significantly from adolescents with overeating without LOC. In a study including children and adolescents (mean age=12.7±2.8y), youth with LOC eating regardless of episode size had significantly greater disordered eating attitudes, emotional eating, eating in the absence of hunger, and depressive and anxiety symptoms, compared to those with either overeating without LOC or no aberrant eating (181).

### **Qualitative features of LOC eating in youth**

Studies of child and adolescent LOC eating suggest that while some aspects of LOC eating in youth are distinct from adult BED (e.g. episode frequency) these constructs are phenomenologically similar. Youth with LOC eating tend to experience less frequent LOC episodes compared to adults with BED. Among community adolescents (12 - 16 years) with LOC, mean episode frequency over the course of one month was  $3.83 \pm 4.46$ , with a range of 1 - 28 episodes (79). The mode number of episodes reported by youth per month was 1. Of all adolescents who reported LOC, most (9.3% of entire sample) experienced only subjective bulimic episodes, some (4.8% of sample) experienced objective bulimic episodes only, and 2.6% of teens experienced both types of episodes. In a clinical sample consisting of 126 overweight, treatment-seeking adolescents ages 12 – 17 years, the mode number of episodes

was also 1, with a range from 1 - 20 episodes. In this sample, most youth experienced objective bulimic episodes ( $n = 26$ ), 13 adolescents experienced only subjective bulimic episodes, and 8 adolescents reported experiencing both types of episodes (162).

Tanofsky-Kraff and colleagues queried 445 youth (8 - 18 years) about their experiences during LOC episodes, and found a number of features in common with traditional BE episodes reported by adults. Specifically, episodes characterized by LOC were associated with eating “forbidden foods,” eating when not hungry, eating alone, having a sense of secrecy around the eating episode, experiencing negative emotions before and after the eating episode, a sense of “numbing” while eating, and feelings of shame or guilt after the episode. Cluster analysis was used to illuminate a subgroup of children subsequently identified as an “LOC eating” cluster based on common features. These children tended to experience episodes characterized by lack of hunger prior to eating, experiencing a “trigger” or negative emotions prior to eating, watching TV while eating, experiencing a sense of secrecy or numbing while eating, experiencing negative emotions after eating, and reporting uncertainty regarding the amount of food consumed. Episodes were often described as a “snack.” Seventy-five percent of cluster members were treatment-seeking, 80% were overweight, 80% were female, 93% were Caucasian, and about half (47%) met full- or sub-threshold criteria for BED.

### **Health-related and psychological correlates**

Among youth, LOC is associated with overweight status, elevated eating pathology, and general psychological symptoms. Similar to adults with BED, LOC eating is associated with greater depressive and anxiety symptoms and elevated restraint, eating, shape and weight concerns (75; 181; 206). Youth with LOC eating also report

greater ineffectiveness and more negative self-evaluation, greater externalizing problems and more problems within parental relationships including under involvement and critical comments about shape and weight (87; 206).

Prospectively, LOC eating predicts increased weight and fat gain (214). Over a  $4.5 \pm 1.9$  year follow up period, children ages 8 - 13 years reporting LOC at baseline gained an average of 2.4 kg more per year compared to youth without baseline LOC. LOC eating is also associated with worsening eating pathology over time. Children with baseline LOC were over five times more likely to manifest full- or partial-syndrome BED an average of nearly five years later (210), compared to those without baseline LOC.

Similar to adult BED and childhood BE, LOC appears to be equally common among racial and ethnic minority populations and Caucasians (206). Among adolescents, LOC was found to be more common among girls compared to boys (79), but this sex difference was not observed among two childhood samples (78; 206). LOC eating is positively correlated with weight status, with the highest rates of LOC seen among overweight, treatment-seeking populations (50; 78). Despite the prevalence and untoward correlates and consequences of LOC, the etiological, momentary processes underlying the mechanism by which LOC eating is linked with excessive weight gain have not been studied among a sample of overweight adolescent girls reporting LOC.

### **INTERPERSONAL MODEL OF LOC**

One potential mechanism for explaining the onset and maintenance of LOC eating is the interpersonal model. The interpersonal model of LOC eating (Figure 1) proposes that difficult interpersonal interactions and relationships, such as conflict, loneliness or



poor communication, lead to negative affect, which in turn results in LOC eating (212).

Thus, there are three key components of the interpersonal model: (1) interpersonal problems, (2) negative affect, and (3) LOC eating. Based on this model, negative affect is the mediating variable between interpersonal problems and LOC eating.

### **Interpersonal problems and negative affect**

The interpersonal model proposes that interpersonal problems lead to negative affect which in turn leads to LOC eating. Thus, the first link proposed by the model is that interpersonal factors are related to and predictive of negative mood. Theoretical perspectives, as well as data from studies examining relationships between interpersonal factors and mood-related outcomes, lend support to a link between these two model components.

### ***Theoretical underpinnings***

The association between interpersonal relationships and mood is grounded in several distinct theoretical perspectives, including an evolutionary perspective (52; 130), Harry Stack Sullivan's *Interpersonal Psychiatry* (202), and John Bowlby's *Attachment Theory* (27), which collectively underscore the importance of interpersonal factors in psychological outcomes.

### ***Evolutionary theories***

Evolutionary theories of the importance of interpersonal relationships for survival assert that physiological responsiveness to interpersonal distress is biologically determined and genetically "programmed." Consistent with data suggesting that interpersonal experiences elicit biological responses, theories of "social pain" suggest that humans are biologically adapted to experience physiological distress in response to

social isolation. Social pain is defined as “the distressing experience arising from the perception of actual or potential psychological distance from close others or a social group” (52). Theories of social pain are grounded in the necessity of social contact for human survival, which is especially true during a child’s first several years of life. It is hypothesized that pain mechanisms involved in detecting and preventing physical danger are mirrored by a “social attachment system” to detect and prevent social separation. Because estrangement from the social group posed a threat to survival throughout evolutionary history, the response of physical pain to distressing interpersonal events is thought to have evolved in order to aid mammals in ameliorating threats to inclusion (52; 130).

#### *Interpersonal theory*

Harry Stack Sullivan (1953) was one of several early scholars who identified the importance of interpersonal context in psychological functioning. Sullivan’s ‘Interpersonal Psychiatry’ denoted the perspective, described below by Mufson and colleagues (140), that mental illness in large part derived from inadequacies in social domains:

Psychiatry is the study of interpersonal relationships under any and all circumstances in which these relationships exist. Sullivan states in his interpersonal theory of emotions that interpersonal behavior of other individuals forms the most significant class of events and objects that trigger emotions in people. He states that a large part of mental disorders results from and is perpetuated by inadequate communication.

Sullivan's emphasis on interpersonal context in the development of psychopathology contributed to the basis for current interpersonal conceptualizations of psychological functioning.

A second root of current interpersonal theory is *Attachment Theory* (27), citing the importance of early interpersonal bonds, and the loss of such bonds, as giving rise to emotional outcomes. Attachment theory describes the crucial role of early relationships in shaping subsequent interpersonal relationships, expectations, and behavior. A core idea proposed by attachment theory is that humans need and form strong affectionate bonds with one another and that the qualities of earlier bonds shape expectations for, and actual, subsequent bonds.

A commonality across evolutionary, interpersonal, and attachment theories is the emphasis placed on interpersonal relationships in shaping emotional outcomes. A contemporary model that extends the work of Sullivan, Bowlby and others, and proposes a therapeutic strategy (interpersonal psychotherapy) that follows the interpersonal conceptualization, is the interpersonal theory of depression, originally proposed by Klerman and Weissman (113). Techniques proposed by Klerman and Weissman's model are consistent with previous interpersonal conceptualizations, and target interpersonal areas of functioning such as communication, interpersonal problem solving, and finding ways to derive greater satisfaction from interpersonal relationships in order to improve emotional well-being. The interpersonal conceptualization of depression does not assume that the entire etiology of depression derives from interpersonal problems, but rather asserts that depression emerges within an interpersonal context (140). Among adults, interpersonal psychotherapy (IPT), based on interpersonal theory, has received



robust empirical support for depression and for preventing depression recurrence. This treatment has therefore been adapted for depressed adolescents by Mufson and colleagues (140).

Interpersonal therapy for depressed adolescents (termed IPT-A) focuses on current interpersonal issues that are relevant to the adolescent, especially those that occur within the adolescent's family (140). IPT-A proposes four specific interpersonal problem areas that relate to adolescents' emotional well-being. Interpersonal problem areas include interpersonal deficits, role disputes, role transitions, and grief/loss. Interpersonal deficits occur when the adolescent lacks skills or strategies to navigate interpersonal relationships. Interpersonal deficits interfere with the adolescent's social functioning in ways such as ability to seek support or establish friendships. The second interpersonal problem area is role disputes, characterized by significant conflict within one or more important relationships. The third interpersonal problem area is role transitions, defined as difficulty adjusting to a new situation such as transitioning from middle to high school, moving, or entering puberty. Significant overlap between interpersonal role dispute and role transition is common, for example, entering adolescence (role transition) is accompanied by a shift in the adolescent's role (role dispute) within the family (e.g. toward greater autonomy). Finally, the fourth interpersonal problem area, grief/loss, is reserved specifically for adolescents experiencing the loss of a significant interpersonal relationship.

#### *Adolescents' interpersonal contexts*

Interpersonal problem areas provide one model for understanding interpersonal functioning among adolescents. Within interpersonal problem areas, conflict or deficits

may occur across one or more relationships, adding a layer of complexity in understanding adolescents' interpersonal functioning. For example, one adolescent's role dispute may occur within her relationship with her mother while another adolescent's role dispute may involve her father. Adolescents' interpersonal contexts include relationships with parents, siblings, extended family members, non-familial adults, peers, romantic partners, and others. Each adolescent's interpersonal network is likely to be highly idiosyncratic.

### ***Empirical Support***

Support for an interpersonal conceptualization of mood is also supported by empirical studies of both peer and parent relationships, which suggest that interpersonal functioning is central to emotional outcomes among adolescents.

#### ***Peer relationships***

The developmental period of adolescence is generally characterized by increasing importance of peer relationships (71; 195). Peer acceptance and friendship are especially salient to adolescents' developing sense of self and psychosocial adjustment. Conversely, deficits in interpersonal relationships with peers, including rejection, stigmatization, and isolation, have been shown to be associated with a variety of psychological problems.

Illustrating the importance of interpersonal factors during adolescence, peer rejection and negative peer relationships are associated cross-sectionally and prospectively with elevated internalizing symptoms. A number of studies have found that relationship problems, including lack of dyadic friendships, peer victimization, and peer rejection, are associated with low self-esteem among teens (23; 128). Controlling

for the role of dyadic friendships and peer rejection, isolation from a social group (“clique isolation”) in early adolescence (ages 11 - 13) was also found to be predictive of the development of depressive symptoms at age 14 years (239). Similarly, among a small sample of youth, lower levels of perceived peer acceptance in 4<sup>th</sup> and 5<sup>th</sup> grade were predictive of later symptoms of dysphoria in 11<sup>th</sup> and 12<sup>th</sup> grade (112). Brendgen and colleagues (2010) assessed youth’s trajectories of depressed mood throughout adolescence, and found that compared to youth without friends, those with non-depressed friends had less elevated trajectories of depressed mood (28).

Among severely impaired teens, peer factors are similarly highly relevant to emotional well-being. Among adolescent inpatients hospitalized for suicidality, severity of suicidal ideation was predicted by interpersonal factors including higher levels of peer rejection and lower levels of close friendship (159). In a separate sample of adolescent psychiatric inpatients, lower levels of peer attachment were predictive of greater depression and suicidal ideation among girls, but not among boys (45).

Bolstering the association between relationships and mood are data indicating that interpersonal psychotherapy is effective in decreasing negative affect and improving interpersonal and social functioning in youth (141; 169; 243; 244). In preliminary studies, psychotherapy targeting the interpersonal context of depression was effective for mood symptom reduction (141).

The impact of interpersonal problems on adolescent functioning is also highlighted by associations between problematic peer relationships and a variety of externalizing problems. In a longitudinal study among youth followed for seven years beginning in 5<sup>th</sup> grade, rejected youth, as compared to those considered popular, average,

or neglected, were found to be at greater risk for negative outcomes such as truancy, suspension, and dropping out of school, throughout a 7 year follow-up interval (121). Similarly, lack of peer acceptance among 4<sup>th</sup> graders was associated with lower academic performance in early adolescence (67).

### *Parental relationships*

Despite the increasing importance of peer relationships in adolescents' social networks, parental relationships are thought to retain high salience in adolescents' psychosocial functioning. Adolescent-parent relationships are thought to be complex and vary across a number of dimensions including warmth, supportiveness, influence, autonomy promotion, behavioral control, and others.

A number of aspects of the adolescent-parent relationship are relevant for adolescents' emotional well-being, behavioral outcomes, and functioning in peer relationships. In studies that examined adolescent-parent interactions in a laboratory paradigm, adolescents' demonstrations of autonomy and relatedness in discussions with their parents was related to superior outcomes two to three years later, including improved self-esteem, superior ego-development, fewer clinician-rated depressive symptoms, and fewer adolescent-reported externalizing problems (5; 6). Similarly, adolescents who experienced more disagreements with their parents at baseline and throughout a seven-year follow-up interval had greater increases in internalizing symptoms, and were more likely to develop an anxiety or depressive disorder at follow-up (171).

Notably, adolescents and parents may differ in their perceptions of the quality of their relationships and interactions. Among 167 young adolescents, McElhaney and

colleagues found that parent reports of their influence on their teens tended to capture the construct of behavioral control, whereas adolescent reports of parental influence tended to capture qualities of warmth and supportiveness. Further, when parents rated their level of influence as high (reflecting overt control), adolescents demonstrated less autonomy and relatedness, and were less likely to seek support in observed interactions across several relationships including mothers, fathers, and friends (135).

#### *Salience of interpersonal relationships among overweight adolescents*

Interpersonal difficulties may be especially salient for overweight adolescent girls. Overweight youth tend to report more frequent teasing, social isolation, and generally compromised interpersonal functioning compared to non-overweight peers (89; 153; 198). In a large ( $n = 90,118$ ) nationally-representative cohort of adolescents ages 13 - 18 years, overweight adolescents received fewer friendship nominations by their peers, suggesting greater social isolation and increased likelihood of being on the periphery of social networks (198). Overweight teens also had increased odds of receiving no friendship nominations compared to normal weight individuals. Among high school students, overweight boys were more likely to experience overt victimization, and overweight girls were more likely to experience relational victimization, compared to their normal weight counterparts (153). For girls, overweight status was also associated with decreased likelihood of dating (153).

#### *Stigmatization of overweight youth*

Teasing and stigmatization are common problems disproportionally faced by overweight youth. In a study examining appearance-related teasing among 10-14 year old children, overweight youth were more likely to be teased about their appearance,



were teased more often about their appearance, were more likely to experience teasing about stigmatizing content (being overweight), and were more upset by teasing, compared to non-overweight youth (89). Teasing of overweight youth was more likely to be perpetrated by peers in general rather than a specific peer. Further, overweight adolescents reported poorer functioning in the social domain of quality of life as compared to their non-overweight peers (63; 178). In summary, given high rates of social stigmatization and teasing experienced by overweight youth, the interpersonal model may be of particular relevance among this population.

### **Negative affect and LOC**

#### ***Theoretical underpinnings***

The second link proposed by the interpersonal model posits that negative affect is associated with and predictive of LOC eating episodes. In cultural milieu, negative mood has long been suggested as an antecedent to overeating and/or BE. Consistent with this perspective are affect theories of BE, suggesting that negative mood states trigger episodes of uncontrolled eating in order to escape from, or alleviate, adverse emotions (88; 109). Affect theories describe mechanisms which may account for the association between negative affective states and subsequent BE. Psychologically, BE has been suggested to temporarily reduce negative affect by serving as a coping strategy (15) or as an escape from self-awareness (90). Arnou and colleagues suggest that binge eaters may use eating to cope with negative emotions. Similarly, Heatherton and Baumeister suggest that the act of BE enables individuals who binge eat to shift from higher to lower levels of self-awareness, allowing an escape from painful or threatening stimuli that otherwise enter awareness. Thus, BE reduces the content of one's experience to physical stimuli

and sensations, allowing an escape from emotional threats and worries (90). Via these mechanisms, negative emotions may serve as perpetuating factors for overeating and obesity.

### ***Empirical underpinnings***

#### ***Self-report studies***

One method for understanding the interplay between negative affect and eating behavior is examining the relationship between self-reported likelihood of eating in response to emotions, termed “emotional eating” and reports of BE. In studies of overweight adults, emotional eating as assessed by self-report questionnaires was found to be associated with presence of BE across studies (53; 132; 165; 240). Among a sample of BED patients only, a significant positive correlation was found between emotional overeating and frequency of binge episodes (132). Similarly, among youth, LOC eating is often associated with the use of food to cope with negative affect. Self-report studies suggest that children with LOC eating have higher trait levels of depression and anxiety (204) and report experiencing negative emotional states prior to engaging in such episodes (207). In a self-report study of emotional eating among a sample of non-treatment seeking children and adolescents, emotional eating was found to be common in response to anger, anxiety, frustration, and depression (211). Over half of children reported occasions of emotional eating. In a non-clinical sample of adolescent girls, emotional eating was found to be correlated with symptoms of depression and anxiety (228). Among overweight treatment-seeking adolescents, adolescents with full-syndrome BED had higher reported negative affect and trait anxiety compared to non-BED youth, and adolescents with recent or past binge or LOC eating had greater depression, anxiety,

and internalizing and externalizing symptoms compared to those without LOC (73). In a separate study, adolescents ages 13 - 17 who reported LOC regardless of the episode size similarly reported significantly higher levels of depression compared to non-LOC youth (77).

#### *Laboratory studies*

Laboratory studies also suggest that negative affect may act as a precipitant of binge episodes among adults with recurrent BE (3; 80; 219). In a laboratory study involving negative or neutral mood induction among adults with BED and non-eating disorder subjects, self-reported mood did not predict food intake during a multi-item food buffet (219). However, reported negative affect was associated with meals labeled as binges and involving LOC (219). In another study involving measured intake of comfort food (chocolate) among 40 obese female binge eaters assigned to either a sad or neutral film induction, Chua and colleagues found that women in the negative mood condition ate significantly more than those in the neutral condition (35). Consistent with adult studies, in a child study involving a laboratory test meal, LOC youth reported worse mood prior to eating than those without LOC (208).

Less is known about the influence of positive mood on eating behavior. One study among 16 - 45 year old women examined the influence of positive mood induction on subsequent intake of a comfort food (chocolate chip cookies) in a laboratory setting (226). Following a comedy clip, women who exhibited a controlled eating style tended to eat fewer chocolate chip cookies in response to the positive mood induction. By contrast, among 'uncontrolled eaters,' viewing the positive mood induction was



associated with slightly greater cookie consumption (226). Additional studies are needed to elucidate the influence of positive affect on intake, especially among adolescents.

#### *Ecological momentary assessment studies*

A number of studies using ecological momentary assessment (EMA) further support the role of negative mood in predicting BE. Among a sample of women with and without BED who monitored their eating and mood for six days using handheld computers, predictors of binge episodes differed based on BED status (80). Among those with BED only, low mood predicted episodes of binge eating (80). In a separate sample of adult women with BED, negative mood was found to be higher prior to binge episodes compared to non-binge times, and negative affect was greatest following BE (194). Similarly, among women with both BED and BN, mood was reported as more negative prior to binge episodes than at regular meal times and random assessments (95). In a non-clinical sample, college women with BE behavior reported significantly greater depression, anger and guilt/self-blame on days that binges were reported compared to non-binge days (233). When mood trajectories surrounding BE episodes were examined, there was no evidence for significant increases in negative affect immediately prior to binge episodes.

In the only pediatric study that used EMA to examine the relationship between negative mood states and eating behavior, there was insufficient evidence to suggest that negative mood significantly contributed to LOC episodes among youth ages 8 - 13 years (94). However, the LOC inclusion criterion for this study was report of one LOC eating episode in the three months prior to study participation, and it is possible that affect-based models of LOC are less relevant for youth with infrequent LOC eating episodes.

## **Full interpersonal model**

### ***Empirical support***

Because interpersonal relationships and social functioning are of vital importance to adolescents (140), difficulty in these domains is likely to produce negative affect, which may trigger LOC eating, consistent with the interpersonal model. In the single study that has examined the validity of the interpersonal model among youth to date, 8 - 17 year old youth of all weight strata completed self-report assessments of interpersonal problems, negative affect, and LOC eating, after which structural equation modeling was used to examine relationships between variables. Study findings (Figure 2) suggested that the interpersonal model was a good fit to the data, with negative affect mediating the relationship between social problems and LOC eating (54).

Also supporting the interpersonal model, preliminary data from a pilot study suggested that reducing LOC eating via interpersonal psychotherapy resulted in weight maintenance in adolescent girls at high-risk for adult obesity (213). In this study consisting of 38 girls who were above-average weight with and without LOC eating, girls with LOC at baseline who received interpersonal psychotherapy were more likely to reduce LOC episodes compared to girls who received health education. Among all girls in the study with and without LOC, girls in the interpersonal psychotherapy group were more likely to gain less than the expected amount of weight at a one-year follow-up visit (213).

LOC eating may be an ineffective emotion regulation strategy over time for several reasons. First, among adults with BE, naturalistic data suggest that negative affect increases after a binge, and thus, relief from negative mood is likely to be fleeting

(42). Similarly in youth, in a laboratory meal study controlling for respective pre-meal affective states, LOC youth reported significantly greater negative affect after eating, including having higher post-meal anxiety, confusion, and fatigue, compared to non-LOC youth (208). Finally, weight gain has been shown to be a long-term consequence of LOC eating (214), suggesting that individuals with LOC are consuming too much energy over time, possibly due to LOC episodes. Because undesirable feelings about shape and weight are a potential source of negative affect (228), weight gain resulting from LOC eating may induce further negative affect and LOC, perpetuating a cycle of negative affect and LOC eating.

## **MEASUREMENT OF AFFECT**

Measurement of affect in any population is limited by the ability of participants to know and report how they feel, a limitation that may be especially salient among individuals with eating pathology. Data among adults suggest that, compared to non-BED obese controls, those with BED may have greater difficulty recognizing and reporting their emotions (33; 41; 157), the clinical term for which is alexithymia.

### **Alexithymia**

Alexithymia is defined as difficulty in identifying and describing feelings and a concrete and externally oriented thinking style (218). Among obese adults, clinically significant alexithymia was more prevalent among those with, compared to those without, BED (41). Among a sample of BED patients only, presence of alexithymia was associated with the severity of the disorder (33). Similarly, in a sample of women only, BED subjects exhibited higher alexithymia scores compared to non-BED women (157).

Although no study has yet examined the construct of alexithymia among youth with LOC eating, adult studies support the possibility that youth with LOC may similarly have difficulty identifying and reporting their emotions. Supporting the possibility that youth with LOC are less able to identify and report emotions, among a large sample ( $n = 445$ ) of 8 - 18 year-old interviewed about their eating episodes, those with eating episodes characterized by LOC were more likely to experience a sense of “numbing” while eating (207).

### **Utility of physiological index of affect**

Difficulty in self-report of emotions suggests the utility of physiological indices of negative affect. Novel assessment strategies using dynamic biomarkers provide an alternative inroad to individuals’ emotional states by capturing the physiological component of human emotions.

## **HUMAN EMOTIONS AND PHYSIOLOGY**

Human emotions are thought to reflect a complex interplay between cognitive processes, physiologic changes, and behavior (13). The experience of emotions is associated with varying degrees of physiological arousal, mediated by neuroendocrine factors, including the hypothalamic-pituitary-adrenal (HPA) axis and the autonomic nervous system (ANS). c

### **Hypothalamic-pituitary-adrenal axis**

#### ***Structure and function***

The HPA axis, one of the body’s primary systems for responding to stress, includes the hypothalamus, the pituitary gland, and the adrenal glands, as well as the

complex interactions and feedback systems that occur among these organs and glands. The hypothalamus and pituitary glands are both located in the brain. The hypothalamus is considered the “master gland” of the endocrine system given its pervasive impact on the pituitary and other endocrine glands. Positioned centimeters above the pituitary in the brain, the structure and location of the hypothalamus and pituitary glands are conducive to their functional relationship. These two structures are connected by a small “stalk,” known as the median eminence, containing nerve fibers and glandular tissue by which nervous and endocrine signals are transmitted to the pituitary gland.

Each adrenal gland is triangular and located superior to each respective kidney. The adrenal glands are composed of two distinct layers, including medulla and cortex. The adrenal cortex is the layer of primary interest with regard to the HPA axis, and produces a group of hormones known as corticosteroids, deriving from the precursor molecule cholesterol. The cortex is subdivided into three layers, producing different hormones as a function of the enzymes present in each layer. The zona fasciculata is the major target of hypothalamic-pituitary hormones, secreting cortisol, the primary downstream product of HPA activation. The adrenal medulla is functionally related to the autonomic (sympathetic) nervous system, secreting epinephrine and norepinephrine in response to sympathetic stimulation. Thus, secretions from the adrenal medulla have nearly the same impact as direct stimulation of the sympathetic nervous system in many parts of the body.

*Mechanism of the HPA axis.* The HPA axis exerts its effects via a number of endocrine signaling pathways whose downstream product is cortisol, responsible for bodily response to stress. This process is initiated when the hypothalamus produces

corticotrophin releasing hormone (CRH), a peptide hormone that descends the median eminence to the pituitary gland to stimulate pituitary release of adrenocorticotrophic hormone (ACTH). In turn, ACTH is released by the pituitary, and targets the fasciculata layer of the adrenal cortex to produce cortisol and corticosterone, as well as small amounts of androgens. An important feature of the HPA axis is the ability of the downstream product, cortisol, to provide feedback to the upstream glands involved in its production. Cortisol has a direct negative feedback effect on both the hypothalamus and the anterior pituitary gland, reducing CRH and ACTH production, respectively. Each negative feedback loop protects against excessive cortisol release.

*Cortisol.* Cortisol has widespread effects throughout the body. One of its most important functions is metabolic regulation and management of stress, which takes a variety of forms. Stress is collectively defined as any threat to the body's state of homeostasis. Stressors can be physical (e.g., hunger, predatory) or mental/emotional (e.g., loneliness) in nature.

One of cortisol's prominent roles in management of acute stress is stimulation of gluconeogenesis, the process whereby complex stored or ingested macronutrients are metabolized into simple forms so that they may be utilized for energy. For example, in the liver, cortisol activates the enzymes required to convert amino acids into glucose and enables mobilization of amino acids from extrahepatic tissue. Similarly, cortisol generally reduces the rates of protein and fat synthesis and storage, and stimulates breakdown of these molecules to be used for glucose formation and energy.

#### ***HPA axis and the interpersonal model***



Although cortisol release is adaptive for managing stressors of a physical nature, it is less well suited for ameliorating stressors of other types. However, because the body's stress response is not well differentiated across various types of stressors, cortisol release is common in response to interpersonal, emotional, and mental stress, in addition to those of a physical nature.

### *Interpersonal events*

Amassed data from laboratory and naturalistic studies suggest that adolescents and adults experience HPA activation in response to interpersonal events. Physiological stress reactivity has been observed in response to situations involving social evaluation (25; 146), interpersonal rejection (201), loneliness (47), and being alone (1; 47). Among adolescents in the natural environment, trait loneliness was associated with blunted diurnal cortisol curves and momentary loneliness was associated with temporary increases in cortisol among youth with high chronic interpersonal distress (47).

Physiological response to interpersonal stress may vary as a function of individual characteristics, with several studies suggesting that girls may be more sensitive to interpersonal stress than boys (146; 170; 200). In a study examining cortisol reactivity to a laboratory interpersonal challenge among girls and boys ages 11 - 16 years, girls' cortisol reactivity to an interpersonal challenge was no different from boys', but among the girls only, greater physiologic response was predictive of self-reported internalizing symptoms (146). Among adults randomized to either an 'achievement' or 'rejection' laboratory stressor, women exhibited greater cortisol responses to the 'rejection' stressor whereas men reacted more strongly to the 'achievement' condition (200).

### *Affect*

Examinations of short-term mood fluctuations using EMA suggest that salivary cortisol may fluctuate as a function of momentary negative affect (1; 186). Adam and colleagues examined adolescents' self-reported mood and salivary cortisol seven times per day for two days, and found that greater self-reported worry/stress and anger/frustration were associated with within-person increases in cortisol (1). Among adults queried in their natural environment, momentary negative affect was associated with higher salivary cortisol and conversely, momentary positive affect was associated with lower cortisol levels (186).

#### *Binge and LOC eating*

Although existing studies suggest that cortisol may be associated with greater food intake among non-eating disordered men and women (60; 217), no study has specifically examined eating behavior in response to stress-induced cortisol secretion among individuals with BED. One recent adult study contrasted cortisol response and desire to binge eat following a cold pressor test among obese women with and without BED (74). Findings suggested that women with BED experienced non-significantly ( $p=.06$ ) greater cortisol response after the stressor compared to non-BED women. Women with BED also reported greater hunger ratings and desire to binge eat, however the intensity of cortisol response was not significantly associated with these factors (74).

### **Autonomic nervous system**

#### *Structure and function*

The second prominent component of the body's stress response is the autonomic nervous system (ANS). The ANS is comprised of the sympathetic and parasympathetic nervous systems which function antagonistically to regulate the body's internal milieu.

In general, the sympathetic component of the ANS is excitatory and functions to respond to challenges, including those of an emotional nature. The sympathetic nervous system typically predominates in times of stress. The second component of the ANS, the parasympathetic nervous system, promotes functions associated with growth and restoration, predominating during times when external stressors are absent. Ability to respond appropriately to stressors, through sympathetic and parasympathetic modulation, is thus critical to adaptive emotional responding.

### *Mechanism*

Parasympathetic and sympathetic components of the ANS influence the cardiovascular system via innervation of the sinoatrial node of the heart. The sinoatrial node is known as the “pacemaker,” generating action potentials that are transmitted throughout the heart muscle, producing coordinated myocardial contractions that constitute the heartbeat. Consistent with its role in responding to environmental stress, the sympathetic nervous system has an excitatory influence on the sinoatrial node, resulting in a more rapid heartbeat. By contrast, the parasympathetic has an inhibitory influence on the sinoatrial node, slowing the heartbeat, which is consistent with its role in lessening physiological arousal to promote restoration.

The sympathetic and parasympathetic nervous systems utilize different signaling mechanisms (neurotransmitters) in communicating with the sinoatrial node. Sympathetic innervation of the sinoatrial node is mediated by release of norepinephrine, whose peak effects are produced approximately 4 seconds following release, with a return to baseline occurring approximately 20 seconds after release (13). Parasympathetic action is mediated by the release of a different neurotransmitter, acetylcholine, whose peak effects

occur about 0.5 seconds following release and terminate within 1 second. Thus, changes in the heart rate mediated by the sympathetic nervous system are slower and longer lasting compared to those mediated by the parasympathetic nervous system.

#### *Measurement of ANS activity via heart rate variability (HRV)*

The difference in oscillatory frequency produced by parasympathetically- versus sympathetically-mediated fluctuations in beat-to-beat intervals enabled the development of indices, collectively known as HRV, to quantify autonomic contributions to heart rate. Defined as the variability in beat-to-beat intervals, HRV indices reflect the degree to which cardiac activity is modulated by sympathetic and parasympathetic components of the ANS (158). Given the importance of the ANS system in emotional responding, such methods provide a non-invasive inroad to the physiologic component of experienced emotions. In general, ability to regulate heart rate to the demands of a situation (e.g., greater variability in the heart rate across situations, “heart rate variability”) is considered an index of humans’ capacity for adaptive emotional responding (158).

#### *Respiratory Sinus Arrhythmia*

A salient factor in influencing parasympathetically-mediated heart rate oscillatory frequency is the respiratory sinus arrhythmia, defined as the rhythmic oscillation in heart rate that co-varies with respiration. Breathing air into the lungs (inhalation) temporarily shunts vagal innervation to the heart, producing an increase in heart rate, whereas exhalation restores parasympathetic influence, decreasing the heart rate, thereby producing a rhythmic oscillation in heart rate that co-varies with each breath. Because the respiratory sinus arrhythmia produces high-frequency fluctuations, oscillations produced by the parasympathetic nervous system, mediated via acetylcholine, are rapid

and short-lasting, and the beat-to-beat oscillations consistent with the respiratory sinus arrhythmia are commonly accepted as an index of parasympathetically-mediated HRV(158).

### ***Clinical relevance***

HRV is a highly relevant clinical biomarker, given its predictive value in cardiac and other-cause morbidity and mortality (44; 122; 225). One mechanism by which HRV is linked with cardiac mortality is via increased likelihood for ischemia. HRV aberrations characterized by vagal withdrawal may predispose cardiac ischemia in everyday life, especially in response to stress (116). This may be especially important among obese and overweight individuals. Compared to their non-overweight counterparts, obese individuals tend to have lower HRV and greater incidence of autonomic dysfunction (124; 227).

*HRV and Eating Disorders.* Abnormalities in sympathovagal balance, evidenced by a shift toward elevated parasympathetic, and decreased sympathetic power, are evident in eating disordered samples. In studies among patients with AN, autonomic profiles are characterized by decreased sympathetic tone, parasympathetic predominance, blunted withdrawal of parasympathetically-mediated HRV in response to standing, and blunted sympathetic activation after a tilt test (119; 123; 144; 163). Among women with BN, data similarly suggest decreased low frequency (LF) power and increased high frequency (HF) power, suggestive of parasympathetic predominance (110; 167; 229), possibly explained by being in a metabolically deficient state (229).

Findings regarding persistence of autonomic abnormalities after weight restoration among eating disorder patients are mixed. Rechlin and colleagues studied



HRV parameters among AN patients of various weight strata, including patients whose weight fell < 75% average body weight, patients whose weight ranged between 75 – 90% of average body weight, and AN patients who were weight restored. Compared to all other groups, severely underweight (< 75% average body weight) anorexic patients had decreased total power during supine and standing positions, decreased HF power while standing, and a decreased LF:HF ratio during supine position (163). These findings suggested that weight status rather than diagnostic status may be the critical determinant of cardiac autonomic abnormalities in eating disorders. Kriepe and colleagues similarly found that weight restoration produced improvement in autonomic control of heart rate (119). In a sample of BN patients, fasting subjects (but not those who were non-fasting), showed decreased LF power and increased HF power at baseline and on recovery after a mental challenge (230). In a separate sample of BN patients, elevated vagal tone was normalized following 8 weeks of Fluoxetine treatment independent of weight status (167). While the contribution of weight status to HRV aberrations in AN appears to be well-established, factors contributing to the shifts in HRV among BN subjects are less clear.

Two studies investigated autonomic functioning in patients with BE in the absence of compensatory behaviors. Messerli-Burgy et al. studied HRV in response to a mental task among BN and BED patients, in comparison to non-BED obese controls. With similar baseline autonomic indices (LF, HF, total power, LF:HF ratio), BED and non-BED groups demonstrated no differences in patterns of autonomic reactivity and recovery in response to the mental stressor, with both groups experiencing expected increases in LF power, but no respective decrease in HF power, during the task. During



recovery, no significant changes were observed in either group (136). By contrast, in a separate study examining HF-HRV, individuals with BED demonstrated augmented parasympathetic withdrawal in response to a mental stressor compared to non-BED obese control subjects (69).

During eating, HRV is thought to shift toward greater sympathetic and decreased parasympathetic, predominance, but empirical findings are mixed. Lu and colleagues demonstrated post-prandial shifts characterized by increases in the LF: HF ratio and decreases in HF power, at both 30 minutes and 60 minutes following a standardized meal (129). Several other studies demonstrate a similar pattern wherein LF: HF ratio increased post-prandially, suggesting a shift toward sympathetic predominance, but the change was not statistically significant (7; 86; 242). In children, the LF: HF shift was observed. Among youth ages 8 - 17 (mean age of 12.5), LF power was significantly increased, and HF power significantly decreased, following ingestion of a solid meal, whereas no HRV shifts occurred in response to water loading (70).

#### ***ANS and the interpersonal model***

*Interpersonal Events.* Few studies to date have investigated within-person HRV changes based on interpersonal factors in the natural environment. In one study, among otherwise healthy adults with varying degrees of depressive symptoms, HRV was moderated by social context, such that when participants with higher levels of depression were alone, they demonstrated lower HRV and greater negative affect, but not when they were with others (177).

*Trait Affect.* HRV data collected in the laboratory suggest that clinically significant mood episodes are marked by aberrations in autonomic regulation (108; 164;

224). Among a small sample of adolescent girls with major depressive disorder and age-matched controls, adolescents with major depression demonstrated lower high-frequency HRV compared to non-depressed girls, suggesting cardiac vagal dysregulation. In a meta-analysis comprised of 18 studies and over 1,000 adults with and without major depression, depressed individuals were found to have lower HRV, and severity of depression was inversely correlated with HRV (108). Rechlin and colleagues assessed HRV among subjects with major depression, reactive depression with a suicide attempt, and control subjects, and similarly found that subjects with major depression tended to have decreased parasympathetic activity while subjects with reactive depression were no different from controls (164).

*State Affect.* Autonomic influence on HRV in response to specific emotions has been under investigation for several decades. Perspectives regarding the specificity of physiological responses to emotions range from the view that autonomic differences are minimal, varying only on dimensions of arousal and valence, through the perspective that each emotion has a specific, fully differentiated autonomic response pattern (118). Kreibig reviewed 138 studies of autonomic activity during various emotions and found that overall HRV tended to decrease during negative emotions, including anger, anxiety, fear, and sadness in the absence of crying (118).

Consistent with the role of the ANS in responding to stress, stress induction generally results in increases in sympathetic activity and withdrawal of parasympathetic activity. This finding, reflected by decreased HF-HRV and increased LF-HRV following laboratory stressors of varying nature, (e.g., (48; 51; 100; 230)), is relatively robust across age groups and stress-induction paradigms. In an early study using trained recall to

induce positive (appreciation) and negative (anger) emotional states among young and middle-aged adults, McCraty and colleagues found that LF power tended to predominate during the anger state whereas total power was increased during appreciation (134). In a later study employing a happiness- and anger-recall paradigm, Kop and colleagues found that the emotion experienced in response to the task was more salient in terms of HRV changes compared to the hypothesized emotional valence of the task. While the happiness task was associated with an increase in LF-HRV among the entire sample ( $n=20$ ) of healthy young adults, closer examination of findings revealed that experiencing happiness during the task was associated with increased HF-HRV power, whereas feeling frustrated during the happiness induction task was associated with increased LF-HRV power and decreases in HF-HRV from baseline (114). No significant changes in HRV were observed during the anger induction task or in response to emotions reported during the anger induction.

Few studies have examined absolute or relative fluctuations in HRV as they occur throughout the day in the natural environment. One study examined the relationship between experienced positive affect and HRV among patients with coronary artery disease, and found that instances of positive affect were associated with greater HF power and reduced LF power (22). Other aspects of momentary experience, including interpersonal context and worrying, have also demonstrated relatedness to specific patterns of autonomic reactivity. Brosschot et al. investigated the influence of worrying on HRV both during the day and during the subsequent sleep period. Among healthy adults, worry and “stressors” were both associated with decreased HRV, although after controlling for worrying, stressors no longer impacted HRV (30).

*Emotion Regulation.* Short-term changes in HRV in response to stress are generally thought to reflect adaptive responding. Studies involving experimentally-induced stressors among children and adults generally suggest a relationship between physiological response to a stressor and trait measures of adaptive responding. In nine-months-old babies, greater parasympathetic withdrawal in response to a stressor (administration of the Bayley Scales of Infant Development, a psycho-diagnostic testing situation) was associated with fewer behavior problems at three years of age. In middle childhood, youth ages 5 - 13 years enriched for predisposition to mood disorder were assessed at two times: At time 1, the child's autonomic response to a sad film was measured, and at time 2, the child's emotional responding was reported by his/her parent, and his/her depressive symptoms were assessed by a clinician. Findings indicated that autonomic withdrawal in response to the sad film clip at baseline was associated with later adaptive emotional responding as reported by a parent, and fewer depressive symptoms as assessed by a clinician (72). By contrast, among a large sample of youth who were 11 years old at baseline, autonomic regulation was unrelated to concurrent depressive symptoms or depressive symptoms assessed two years later (26). Among adults, Weinstein and colleagues examined autonomic response to a stressor (exercise withdrawal among regular exercisers) and subsequent mood symptoms among 40 women and men who exercised regularly (234). Although exercise withdrawal did not produce autonomic changes within-subjects, reduced HRV at baseline was significantly predictive of development of negative mood in response to the period of exercise withdrawal.

### **Physiological biomarkers as indices of emotional responding**

An accumulation of literature highlights the relevance of physiologic processes in human emotions, however, physiologic response is but one, among many, bio-behavioral components of emotion. The similarities in physiologic response to stressors of a wide variety suggest that the physiological component of emotion reflects a *general activation or arousal*, rather than denoting a specific emotional state (e.g., anger versus anxiety). As a measure of arousal, cortisol and HRV responses have demonstrated relatedness to self-reported affective states as well as aspects of emotion regulation measured longitudinally. Despite that cortisol and HRV do not capture the entirety of one's emotional experience, they may be highly relevant in understanding or predicting behavior related to stress or negative affect, such as LOC eating.

## **ECOLOGICAL MOMENTARY ASSESSMENT**

### **Momentary nature of interpersonal model**

Although sound theory (212) and preliminary retrospective research (54) support the mechanisms by which interpersonal model variables impact excess weight gain in adolescent girls, no study has directly examined the interpersonal model in the natural environment. While the mechanisms proposed by the interpersonal model of LOC have not been well-studied, it is likely that the processes are inherently momentary. The variables not only change over the course of several months, but rather, change over the course of days or minutes (14). A momentary example of this model might be that an adolescent girl has an argument with her mother (interpersonal problem), experiences sadness as a result of this argument (negative affect), and eats while experiencing an inability to control what or how much she is eating (LOC eating) (212). Cross-sectional,



prospective and longitudinal research designs are unable to adequately assess these types of momentary fluctuations. Past research on negative affect and aberrant eating is limited by the inherent drawbacks of traditional self-report assessments. Retrospective recall biases and the necessity for participants to summarize behaviors or experiences over days, weeks or more restrict conclusions that can be drawn from these data. One research methodology that is specifically designed to assess moment-to-moment changes in behaviors and mood that enhances validity in the naturalistic environment is ecological momentary assessment (EMA).

### **Self-report measures**

EMA is an assessment strategy that relies on portable measurement techniques that are applied in “real time” in the participants’ natural environments. The use of EMA avoids concerns of generalizability from laboratory studies by having participants provide data in their naturalistic setting. By using EMA, limitations associated with self-report methodology, including retrospective recall biases and the need for participants to summarize behaviors, cognitions, or emotions over a period of time, are eliminated. EMA data is gathered through the use of small electronic devices, such as palm top computers (180). EMA has been successfully used to study a wide variety of problems including stress and coping (24), cigarette smoking (179), chronic pain (197), and self-injurious behaviors (139). Notably, EMA has been used to study BN (56; 188), AN (59; 156), BED (58; 194), and LOC eating in children (94).

### **Physiologic measures**

With the development of novel ambulatory assessment strategies, momentary physiological changes that accompany stress have become increasingly accessible. Use



of EMA to examine physiologic stress indices in the natural environment has thus expanded, with a number of dynamic biomarkers, including endocrine (e.g., cortisol), cardiovascular (e.g. heart rate, blood pressure), and autonomic (e.g. HRV) indices now able to be assessed using ambulatory equipment. Although these measures specifically denote physiologic, but not cognitive or behavioral, components of emotion, they likely will provide highly relevant information for understanding mood and LOC eating in the natural environment.

Despite more frequent use of cortisol to index physiologic stress in prior studies conducted in the natural environment (e.g. (1; 47)), there are a number of advantages to use of HRV. Methodologically, HRV equipment is applied at a single time and records HRV continuously until the equipment is removed. In contrast, salivary cortisol requires the participant to perform several steps at various sampling intervals. HRV therefore provides a greater span of data and requires less interference in participants' day-to-day life, improving convenience and generalizability to a non-research setting. Clinically, HRV may be of particular relevance to overweight youth given its associations with obesity among adults. Although some studies suggest that women with AN or BN (119; 163), particularly those in a fasting state, experience shifts toward chronic parasympathetic predominance, this has not generally been found among BED samples (136). Rather, BED subjects may experience greater decreases in parasympathetic activity in response to stressors, although such data is preliminary (69). Furthermore, HRV is an important clinical biomarker that has been linked to depressive mood states among adults and youth, and emotional responding across the life span. Given the high prevalence of mood aberrations among BED patients, and the current study's hypothesis

that negative affect plays an etiological role in LOC eating, we suspect that HRV may be a salient index of affect within a sample of adolescent girls with LOC. Also, in light of the hypothesis that LOC eating may serve the function of emotion-regulation, alterations in HRV, thought to comprise emotional responding, may be especially salient for predicting LOC episodes.

## **CHAPTER 2: RATIONALE AND SPECIFIC AIMS**

### **RATIONALE**

Binge and LOC eating in youth are associated with adverse psychosocial correlates and are predictive of negative physical and psychological outcomes including excess weight gain and full-syndrome BED (65; 196; 205; 210; 214). The interpersonal model may provide one explanation for the onset and maintenance of LOC eating (212). Given the importance of social and interpersonal factors during adolescence (140), the interpersonal model may be especially relevant for overweight adolescents who are often the target of negative social interactions.

The current proposal will test the mechanisms associated with LOC eating during adolescence using a naturalistic assessment strategy. Since adolescents with LOC eating may have difficulty identifying and reporting affective states, the present study will also measure HRV as a physiologic index of negative affect.

### **SPECIFIC AIMS**

This study has three specific aims. The first aim is to explore the feasibility of testing the interpersonal model of LOC eating in a temporally-sensitive and ecologically valid manner using two EMA strategies: self-report data gathered through the use of handheld digital devices and physiologic data (HRV) gathered using ambulatory electrocardiography. Self-report data will be collected for all components of the interpersonal model: interpersonal problems, negative affect, and LOC eating.

Physiologic data will be collected as an index of the negative affect component of the

interpersonal model. Based on prior EMA research among children, in which children with and without LOC eating achieved a mean compliance rate of 74% over a study period of 4 days (94), as well as the successful completion of ambulatory monitoring of HRV in the natural environment among adults (e.g. (22; 30; 177)), we expect that aim one will be achieved, enabling exploration of aims two and three. The second study aim is to examine the correlation between self-reported negative affect and heart rate (HR) and HRV. The third study aim is to examine the mediation hypothesis that interpersonal problems precede and predict LOC eating via negative affect.

#### **GENERAL ANALYTIC STRATEGY**

EMA studies involve many repeated assessments, with the number and spacing of assessments varying between subjects (180). The nature of EMA data limits its ability to be analyzed using traditional methods for several reasons. First, EMA data implies a nested design in which random variability derives from two sources: between- subjects and within-subjects. Past strategies for handling multilevel data have including aggregating data and ignoring sampling at the person level (176). Although aggregation can be conducted at the between- and within- subjects levels, it violates the heteroskedasticity assumption necessary for general linear modeling. Specifically, the variance for each individual's mean across aggregated data is dependent on the number of observations collected. Therefore, the variance of aggregated data is actually derived from two sources, typical unexplained variance, as well as variance due to the number of assessments involved in the computation. A second strategy for the analysis of EMA data is to ignore the contribution of between-subjects random error (ignoring sampling at

the person level) (176). This strategy is similarly limited because it may attribute within-subject variability to unmeasured between-subject factors, inflating the estimate of within-person variability.

Repeated-measures analysis of covariance (ANCOVAs) is recommended as the best least-squares approach to EMA data. ANCOVA allows for the creation of a random factor to represent 'person,' enabling all unexplained variance across the person-level to be accounted for. However, ANCOVA are not ideal for EMA data because they require an identical number and timing of observations across each person, and individuals with missing observations are excluded from the analysis (176).

Given that traditional methods are limited in their capacity to analyze unbalanced and multi-level assessments, new procedures were developed to accommodate unbalanced designs that often characterize EMA data (176). Multilevel models accommodate EMA data by including an equation term representing a separate intercept for each individual, accounting for between-subjects variance that is not accounted for by other model factors. Multilevel models accommodate the violation of the assumption of independent observations inherent in traditional regression models. For all models, a subject-specific intercept and a variance components correlation structure were used. All coefficients were treated as fixed. The coefficients of primary independent variables were treated as fixed because models did not converge when effects were treated as random, and because fixed effects produced the most parsimonious model. In models including multiple assessments per person, significant effects would indicate a relationship between independent and dependent variables, but it would not reveal whether the source of the relationship is due to a between- versus a within-subjects level

effect. A significant limitation of multilevel linear models, therefore, is the inability to parse between versus within subjects variability. This will be discussed in further detail in the results section. Statistical Package for the Social Sciences (SPSS) version 20.0 (101) will be used to examine all models including those with continuous and categorical dependent variables.

## **HYPOTHESES AND STATISTICAL ANALYSES**

### **Aim 1**

To evaluate the feasibility and acceptability of using a naturalistic study design to test the interpersonal model of LOC eating among overweight adolescent girls who report LOC eating.

#### ***Hypothesis 1.***

We hypothesize that participants will be able to complete study procedures and that the procedures will be feasible and acceptable to overweight adolescent girls with LOC eating patterns.

Feasibility and acceptability will be assessed according to three dimensions: (1) compliance, (2) laboratory visit attendance, and (3) acceptability questionnaires.

#### ***Hypothesis 1a.***

Participants will be compliant with random recordings, defined as a response rate greater than or equal to 75%, and event-contingent recordings.

In the only existing pediatric study of affective triggers of LOC eating using EMA in the natural environment, Hilbert and colleagues found that among 118 children with



and without LOC, children answered an average of nine out of twelve random phone calls, achieving a compliance rate for random recordings of 74.1% (94). Therefore, we have selected a response rate of 75% to constitute “compliance” in the current study. We expect that adolescents will be more compliant than youth ages 8 - 13 years, per Hilbert et al. (94). Rates of compliance with EMA random recordings will be calculated according to the total number of random EMA recordings divided by the total number of EMA recordings.

*Hypothesis 1b.*

Participants will attend all laboratory visits required for collection of EMA data.

Laboratory visit attendance will be calculated to assess the feasibility of attending multiple visits required to conduct studies using EMA and Holter monitoring procedures. Visits will be counted as either (a) attended, (b) canceled-rescheduled, (c) canceled-not rescheduled, or (d) no show. We will track any lost data due to (a) missed visits and/or (b) equipment malfunction between visits. Procedures will be considered feasible if an attendance rate (including rescheduled visits) greater than 75% is achieved.

*Hypothesis 1c.*

The study procedures will be acceptable to overweight adolescent girls with LOC eating.

To assess acceptability, girls will be administered an acceptability questionnaire (Appendix 1). Average rates of acceptability will be calculated for each questionnaire item and for a total score. The procedure will be considered acceptable if a mean rating (across all questionnaire items) greater than or equal to ‘4’ is achieved.

*Hypothesis 1d.*

The association between compliance rates, attendance, and acceptability ratings, and the following factors will be examined: LOC eating, negative affect, interpersonal problems, age, race, and anthropometric variables such as body mass index (BMI;  $\text{kg/m}^2$ ).

Based on prior research that has been conducted in our laboratory (213), we do not expect that LOC eating, negative affect, or interpersonal problems will impede study compliance, attendance of study visits, or acceptability ratings. Further, we expect that race and BMI will be unrelated to participants' compliance rates, attendance, and acceptability ratings. This hypothesis will be tested using three multiple regression models. Each operationalized measure of feasibility (compliance, attendance, acceptability) will constitute the dependent variable in respective models. Independent variables will be entered forward, with demographic variables (age and race) comprising block one, anthropometric variables (BMI) comprising block two, and interpersonal model components (LOC, negative affect, interpersonal problems) comprising block three, in each respective model. Interpersonal model independent variables were derived by aggregating the values for respective variables over the course of the study period.

## **Aim 2**

To evaluate the concordance of self-reported negative affect and autonomic indices HR and HRV among overweight adolescent girls with LOC eating.

### ***Hypothesis 2.***

Self-reported negative affect will be correlated with the following time- and frequency- domain indices of HRV in the following directions: Positive associations are

expected to be found with respect to HR; inverse associations are expected to be found with respect to parasympathetically-mediated indices of HRV; the association between overall HRV and negative affect is an exploratory aim.

Self-reported negative affect will be assessed using the negative affect subscale of the Positive and Negative Affect Scale (PANAS) (125), administered electronically via palmtop computers at both event-contingent and random recordings throughout the two-week study interval. This data collection strategy will produce approximately five EMA recordings of self-reported negative affect on weekdays and eight on weekends.

Physiological measures include HR, total HRV, and indices of parasympathetically-mediated HRV, assessed using time- and frequency-domain analyses (216). Each of these will be explained in further detail in the “methods” section.

A series of mixed-model analysis will be used to examine the relationships between self-reported negative affect and HR and HRV indices, with the within-subjects time variable nested within the between-subjects person variable. HR, total HRV, and indices of parasympathetically-mediated HRV during the five-minute interval prior to the EMA prompt (as described in the “measures” section) will constitute the dependent variable and self-reported negative affect will constitute the independent variable.

Because HRV is a known correlate of obesity in adults (124; 227), BMI will serve as a covariate in these models. Based on prior studies suggesting that individuals experience decreased parasympathetically-mediated HRV during mood episodes (e.g. (224)) and temporary negative affective states (e.g (134)), we hypothesize that parasympathetically-mediated HRV will be inversely associated with self-reported negative affect. All types of recordings: event-contingent, interval-contingent, and random, will be included in

analysis of the relationship between self-reported affect and HRV. To test whether the association between self-reported affect and HRV indices vary as a function of the type of response (random, event-contingent, interval-contingent), response-type will be entered into the model as a covariate. If significant, differences between the self-report and HRV relationship across response-types will be explored.

### **Aim 3**

To evaluate the validity of the interpersonal model positing the mediation hypothesis that interpersonal problems precede and predict negative affect, negative affect precedes and predicts LOC eating, and that negative affect mediates the relationship between interpersonal problems and LOC eating.

#### ***Hypothesis 3.***

Interpersonal problems will precede and predict LOC eating. Interpersonal problems will also precede and predict negative affect, which will precede and predict LOC eating. When negative affect is accounted for, the relationship between interpersonal problems and LOC eating will be attenuated.

Testing a mediation model involves a number of steps (Figure 3) including the following: First, demonstrate that the initial variable (X) is correlated with the outcome variable (Y) (path c). Second, demonstrate that the initial variable (X) is correlated with the mediator variable (M) (path a). Third, while controlling for the initial variable, show that the mediator variable (M) is correlated with the outcome variable (Y) (path b). Finally, to establish the degree to which M is a mediator between X and Y, calculate the degree to which the correlation between X and Y is reduced when controlling for M. Complete mediation is established when the effect of X on Y, controlling for M, is zero.

In the case of the interpersonal model, interpersonal problems represent the initial variable (X), LOC eating represents the outcome variable (Y), and the proposed mediator (M) is negative affect. The following specific hypotheses of Aim 3 correspond to the steps involved in testing mediation.

*Hypothesis 3a.*

Interpersonal problems will precede and predict LOC eating episodes (path c).

Interpersonal problems in the natural environment will be measured using modified questions from the Social Adjustment Scale, Self-Report (235), administered electronically via palmtop computers. LOC episodes will be measured using modified questions from the Eating Disorder Examination (61) to assess feelings of LOC during eating episodes. LOC data will be collected electronically via random and event-contingent reports using palmtop computers carried by the participants.

Mixed modeling will be conducted, with the within-subjects time variable nested within the between-subjects person variable. Interpersonal problems will constitute the independent variable (X) and LOC eating episodes will constitute the dependent variable (Y). LOC will be examined continuously and dichotomously. In analyses of LOC as a continuous variable, indices of skewedness and kurtosis will be examined as indices of normality, and transformations will be applied, if necessary. As a dichotomous variable, “high” LOC episodes include after-meal recordings in which adolescents report a ‘4’ or higher (range 1 – 5, 1 = not at all, 5 = a lot/very much) on any LOC question comprising the LOC composite scale. All other episodes will be categorized as “low” LOC. The intent of using this method is to distinguish eating episodes in which a definite sense of LOC was experienced versus other episodes. Age, race, and BMI will be explored as

covariates. Based on preliminary support for the interpersonal model (12), we hypothesize a positive correlation between interpersonal problems and LOC eating.

*Hypothesis 3b.*

Interpersonal problems will precede and predict negative affect (path a).

Interpersonal problems in the natural environment will be measured using modified questions from the Social Adjustment Scale (235) administered via electronic palm top computer. Self-reported negative affect will be measured using the Positive and Negative Affect Scale PANAS (125). Physiologic indices of negative affect include HR, total HRV and parasympathetically-mediated indices of HRV. Questions from the PANAS will be administered electronically via palm top computers, and HRV indices will be generated from electrocardiogram (ECG) data gathered continuously using ambulatory monitoring. Depending on the degree to which self-reported and physiologic index of negative affect are correlated, they may be combined to create a latent “negative affect” variable.

Two separate random mixed models will be examined with PANAS and physiologic indices (HR, total HRV, and parasympathetically-mediated indices of HRV) as dependent variables in each respective model. Random mixed models will be conducted with the within-subjects time variable nested within the between-subjects person variable. Interpersonal problems will constitute the independent variable, and negative affect (measured by PANAS and HRV indices, respectively) will constitute the dependent variable. A number of potential covariates will be explored including age, race, and BMI. One study using HRV suggested that depressed subjects experienced reduced HF-HRV when alone compared to when with others (177), and a number of



studies suggest that adolescent and adult females experience physiologic stress following interpersonal stressors (1; 47; 201). One adolescent study indicated that girls' physiologic responses to an interpersonal stressor were associated with later internalizing problems (146). Although no study has directly tested links between interpersonal events and negative affect in an ambulatory setting among adolescents, existing literature supports the hypothesis that higher interpersonal problems will be associated with greater negative affect.

*Hypothesis 3c.*

Negative affect will precede and predict episodes of LOC eating (path b).

Self-reported negative affect will be measured using PANAS scores. Physiologic indices of negative affect will include HR, total HRV, and parasympathetically-mediated indices of HRV. LOC episodes will be measured using the Eating Disorder Examination to assess feelings of LOC via random and event-contingent reports using palm top computers carried by participants. Using mixed modeling, time will be nested within subjects. Negative affect, as measured by the PANAS, HR and HRV indices, (and possibly a latent variable created from PANAS and HRV indices, if these constructs are found to be related), will constitute the independent variables in three respective models, and LOC will constitute the dependent variable. LOC will be examined continuously and dichotomously. Age, race, and BMI will be examined as potential covariates.

Based on adult literature citing a role for negative affect in precipitating LOC episodes, it is hypothesized that greater self-reported negative affect will be associated with LOC eating measured continuously and dichotomously. Because greater HR and lower parasympathetically-mediated indices of HRV are thought to be key indicators of

the autonomic stress response, indicative of negative affect, we expect that greater HR and lower parasympathetically-mediated HRV will be associated with LOC eating measured continuously and dichotomously. Because all eating episodes are characterized by changes in HRV (70; 129), the period of HRV data selected for analysis will be that which was recorded 30-60 minutes prior to the eating episode, depending on the duration of the eating episode. By doing so, potential HRV changes that occur in response to eating will be not be subject to analysis.

*Hypothesis 3d.*

Negative affect will mediate the relationship between interpersonal problems and LOC eating. In other words, the correlation between interpersonal problems (X) and LOC eating (Y) will be significantly reduced when controlling for negative affect (M).

Interpersonal problems, negative affect, and LOC eating will be measured as described in hypotheses 3a-3c. The final step in testing for mediation is to examine whether the relationship between the independent variable (X) and dependent variable (Y, Figure 4, path c) is significantly reduced when controlling for the mediator variable (M, path c') (17). If the effect of the independent variable on the dependent variable is no longer significant when controlling for the mediator, the finding supports full mediation. If both the independent variable and the mediator are significant predictors of the dependent variable, the finding supports partial mediation.

There are a number of limitations inherent in the above (17) four step method and therefore some researchers suggest calculating an indirect effect, and then testing for its significance, as a superior method of supporting a mediation model. There are two ways to calculate the value of an indirect mediation effect. Judd and Kenny (1981) suggest

calculating the difference in the correlation coefficient between the models in which X predicts Y, versus that in which X predicts Y controlling for M (105). Alternatively, the Sobel test (189) involves computing the product of the correlation coefficients from models in which X predicts M, and in which M predicts Y, controlling for X. Whichever method is used, testing for the significance of the mediation effect is then performed. Mallinckrodt and colleagues (131) review a number of methods for testing the significance of mediation effects, the most innovative and widely accepted of which include bootstrapping re-sampling procedures popularized by Shrout and Bolger (182) and structural equation modeling. Because the current study is a pilot study with  $n = 10 - 20$ , statistical power for employing such methods is lacking and is not reviewed in detail here.

## **CHAPTER 3: RESEARCH DESIGN AND METHODOLOGY**

### **OVERVIEW**

This investigation was a naturalistic study with the primary aim of evaluating the utility of using an EMA paradigm to test the interpersonal model of LOC eating. The first study aim was to examine the feasibility and acceptability of the proposed methodology. Our second and third aims tested the significance of the interpersonal model.

Primary variables assessed among participants were self-reported interpersonal problems, negative affect, and LOC eating behavior, and a measured index for negative affect, HRV. Self-report variables were assessed for two weeks, an average of three to five times per day. For two days of the protocol, participants completed ambulatory monitoring of HRV via electrocardiography.

### **PARTICIPANTS**

Thirty adolescent females from the community with a BMI at or above the 85<sup>th</sup> percentile for age and sex (151), and who reported engaging in at least two episodes of LOC eating during the month prior to assessment, were included in the current study. We selected to study an overweight population given the increased prevalence of LOC eating among overweight adolescents (50; 148) and because the interpersonal model of LOC may be especially salient for overweight adolescent girls. For this reason, we restricted our sample to adolescent females, as sensitivity to interpersonal distress is thought to be greater among females (170; 200). Finally, we selected to set the minimum

number of LOC episodes for study inclusion at two episodes. Existing EMA studies among adults with disinhibited eating have often examined participants with full-syndrome BED (e.g. (57; 194) or recurrent BE (e.g. once per week for six months, (42)). Because BED is rare among adolescents, we selected a more lenient inclusion criteria compared to the majority of adult studies. Although a prior child EMA report of LOC eating behavior included youth reporting a single LOC episode within the three months prior to assessment (94), an episode frequency of once every three months was viewed as too infrequent to sample the behavior of interest (LOC eating). Therefore, to maximize the likelihood that study participants would experience at least one LOC episode during the study, we selected a two-episode minimum criterion for study inclusion (based on the rationale that if an individual experienced two LOC episodes per month, she would be more likely to experience one episode during a two-week interval).

In order to be included in the present study, participants were required to be English speaking and cognitively capable of completing study procedures. Cognitive ability to complete study procedures was gauged by the participant's ability to understand the consent procedure and complete baseline testing measures (e.g., interviews, questionnaires). This was assessed by the principal investigator of the current study, an advanced graduate student in clinical psychology with experience conducting neuropsychological and developmental testing among children and adolescents. Individuals were excluded (and provided treatment referrals as needed) for presence of an obesity-related medical complication or other major medical illness, use of medications affecting eating or body weight, pregnancy, or presence of a major psychiatric disorder.

Presence of a major psychiatric disorder was determined during a telephone screening and/or by a psychiatric screening interview administered during participants' initial visits.

The study was approved by the USUHS Institute of Child Health and Human Development institutional review boards. Parents provided written consent for study participation, and all girls provided written assent.

## **RECRUITMENT**

Overweight girls, age 12 - 17 years, were recruited from Washington, DC and local suburbs in Maryland and Virginia. Recruitment efforts were targeted toward parents of overweight adolescent girls and included a number of methods found to be effective in prior studies: (1) Letters to area pediatricians, family medicine physicians, and clinics requesting referrals of families with a daughter who is overweight; (2) Advertisements in local newspapers; (3) Flyers (Appendix 2) posted at local facilities (e.g., libraries, supermarkets, fitness clubs) with permission; (4) Flyers posted on free online services such as "Craigslist" and online local news stations; (5) Flyers posted to local middle and high school parent listserves by request; (6) Direct mailings and email contact with families who expressed an interest in past eating-related research studies for adolescent girls at the Uniformed Services University and the National Institutes of Health. Adolescents with whom contact was initiated were ineligible for prior studies for various reasons; and (7) Direct mailings to families in the greater Washington D.C. metropolitan area obtained from a direct marketing services company. Mailings included a brief description of the intent of the mailing (Appendix 3) and a copy of the study advertisement. All advertisements targeted parents and included language indicating that the study aimed to recruit overweight adolescent (12 - 17



years) girls who may experience difficulty controlling their food intake. Advertisements specified that no treatment was provided. These efforts were also highly effective in recruiting participants for prior studies. Interested participants were screened over the telephone to determine potential eligibility. If a girl was eligible, she was scheduled for a screening visit.

## **PROCEDURES**

The study was a two-week observational study using EMA, aimed to examine moment-to-moment associations between participants' relationships and interactions with others, mood, and eating patterns. HRV assessment was conducted on two days of the 14-day assessment period. Due to the preliminary status of HRV as a measure of negative affect for adolescent girls with LOC eating behavior, and due to the burdensome nature of wearing a Holter monitor, we assessed HRV for two days only.

There were three primary components to the study. The first part consisted of a screening visit lasting approximately four hours, occurring on a weekday or weekend morning. If a participant qualified for the study, she was shown how to use the palm top computer at the end of the screening visit. The participant was then provided a chance to practice using the equipment. During the second part of the study, each participant completed a one-day practice period to assess compliance and to ensure that the participant understood the self-monitoring procedures. For the third part of the study, participants carried the palm top computer for two weeks. During this portion of the study, girls returned to the study site 1 - 2 times per week to provide data and obtain new palm top computers. For two days of the 14-day monitoring period, participants' HRV

was monitored. At the end of the two-week EMA assessment, participants returned their equipment and completed evaluation forms. The specific procedures completed at each stage of the study are detailed below:

### **Part 1: Baseline screening appointment**

The baseline screening appointment occurred on either a weekday or weekend in the morning. Girls came to USUHS in a fasted state, having had nothing to eat or drink except for water starting at 10 pm the night before the visit. After fasting measurements were completed, participants were provided with a breakfast. A parent or guardian accompanied their daughter to the first visit. The initial visit lasted approximately four hours. During the baseline visit, families completed the consent/assent process and girls underwent assessment of body composition, including measured height and weight. Girls also completed interviews and questionnaires assessing eating pathology, psychological status, and additional constructs relevant to variables in the interpersonal model (relationships, mood, and eating). The specific measures used are detailed in the 'measures' section. At the end of the baseline assessment, eligible participants were trained on the EMA device.

### **Part 2: EMA practice period**

Following the screening visit, participants practiced EMA data collection for one day. The practice day involved completing the EMA assessments in a manner consistent with the EMA training. In EMA research, participants often find the practice period to be helpful in clarifying the requirements of the protocol (57). If participants were compliant with procedures during the practice period, as defined by demonstrating an understanding of protocol requirements and responsiveness to the majority of random recordings, the

data collection phase of the study was initiated. A second aim of the practice period was to attenuate any reactive effects of the self-monitoring.

### **Part 3: Data collection**

After completing the one-day practice period, the research team uploaded and inspected the adolescents' data and compliance was reviewed. Participants were provided with a new palm top computer for the data collection phase of the study. Over the following 14 days, participants met with the study team approximately one to two times per week to provide data and obtain a new palm top computer. Frequent meetings with the study team ensured that minimal data was lost in the event of equipment malfunction, or in the case that a participant lost or broke the equipment. For compliant participants, collection of physiological data was conducted on two days of the 14-day EMA data collection period.

At the end of the two-week EMA assessment, participants returned their palm top computer and completed evaluation forms. Specifics of the EMA data collection period are described in the 'measures' section.

## **MEASURES**

### **Body composition**

Girls were weighed and measured using a calibrated electronic scale and stadiometer. BMI was calculated from measured height and weight. BMI percentiles and Z-scores were determined using standards established by the 2002 Center for Disease Control growth charts (120; 151). Body composition was assessed using air

displacement plethysmography (Life Measurement Inc., Concord, CA) to determine fat-free mass and fat mass.

## **Interviews**

Semi-structured interviews, assessing girls' psychological status and eating pathology, were administered to screen for study eligibility, psychiatric symptoms, LOC eating, and eating disorder symptoms.

- a. The Eating Disorder Examination (EDE), version 12, with adaptations to assess DSM-IV-TR BED (61). The EDE is considered the gold standard for the assessment of disordered eating pathology, evaluating specific DSM-IV-TR eating disorder diagnoses (8), identifying presence and frequency of binge, LOC and overeating episodes, and assessing disordered eating attitudes and behaviors. Tests of the EDE's discriminant validity, internal consistency, concurrent validity (61) and test-retest reliability (168) support its use in adults and adolescents (73). For the current study, the interview was used to determine the presence of at least two episodes of LOC eating (subjectively large or objectively large binge episodes) during the past month and to exclude adolescents meeting criteria for AN or BN.
- b. Schedule for Affective Disorders and Schizophrenia for School-Age Children (KSADS) (107). The KSADS was used to assess psychiatric status including presence of a major psychiatric disorder warranting study exclusion. The KSADS is a reliable and valid semi-structured diagnostic interview with good interrater reliability that assesses childhood and adolescent diagnoses listed in the DSM-IV-TR (8). The KSADS was also used to operationalize total psychiatric symptoms, and three indices were derived including: (1) "mood/internalizing" symptoms

(comprising symptoms of depression, suicidal ideation, mania, and panic disorder);

(2) “behavioral/externalizing” symptoms (comprising symptoms of attention deficit hyperactivity disorder, oppositional defiant disorder, conduct disorder, and alcohol and drug use), and (3) “Total psychiatric symptoms,” comprising all items.

Symptoms were categorized as absent (score = 0), sub-threshold (score = 1) or present (score = 2), and scale totals were derived by summing items comprising respective scales.

### **Questionnaires**

- a. Beck Depression Inventory-II (BDI-II) (18). The BDI-II is a 21-item self-report measure that assesses depressive symptoms. Single-item responses range from zero to three, with corresponding total scores ranging from 0 - 63. Although the BDI-II is not a diagnostic tool for major depressive disorder, scores from zero to nine are thought to indicate minimal depression; scores in the range of 10 - 16 are thought to reflect mild depression; 17 - 29 may indicate moderate depression; and scores of 30 or greater are thought to reflect severe depression. Psychometric properties among adolescents are established (117).
- b. State –Trait Anxiety Inventory for Children (STAI-C), Trait version (190). The STAI-C, Trait version, is a well validated 20-item measure used to assess trait-level anxiety. Each item is rated on a scale of 0 – 2, with total scores ranging from 0 – 40. Reliability and validity are well established in an adolescent sample (145).

### **EMA**

Self-monitoring of psychosocial variables was recorded on handheld palm top computers (PalmPilot™) using a combination of three types of recordings: (1) random recordings, (2) event-contingent recordings (e.g., whenever a target behavior occurs) and (3) interval-contingent recordings (e.g., at pre-determined times of the day). This combination aimed to ensure comprehensive sampling of the target behavior (e.g., LOC eating) as well as other variables of interest (e.g., negative affect and interpersonal problems). Each assessment strategy is detailed below. The time required to respond to each prompt was approximately 2 - 3 minutes.

### ***Protocol***

- a. Random recordings. Random recordings consisted of a series of questions primarily regarding interpersonal problems and negative affect. Participants were signaled by the palm top computer three to five times per day to complete signal contingent reports. To ensure adequate sampling throughout the day, the entire sampling period (11:00 to 23:00) was stratified into five intervals. Each of the five daily signals occurred in one of these intervals. Signals were randomly distributed around target times of 11:10, 13:50, 16:30, 19:10, and 23:50, each occurring within one of the five intervals. Given our population of adolescents, programming of the palm top computers was tailored to ensure that participants were not signaled during the school day. Therefore, even though the sampling period spans from 11:00 through 23:00, participants were not signaled until after 3 pm between Monday and Friday, resulting in fewer (3) overall signals on these days. Existing research indicating that LOC eating episodes typically occur after school hours in the afternoon and evening suggests that this approach will still reasonably capture most LOC episodes (187;



207). Prior work supports that tailoring the signaling schedule is very effective in facilitating compliance and significantly reduces the burden associated with completing the protocol (94).

- b. Event-contingent recordings. Participants completed event-contingent recordings whenever a target behavior, eating, occurred. Consistent with the definition of an ‘eating episode’ as explained to youth during administration of the EDE (61), ‘eating episodes’ were systematically defined to participants as: “Any time of eating or drinking that you consider to be a meal or snack. If the food or drink was considered an ‘eating episode’ to you, we would like you to report on it. For example, we do not need you to complete a response after taking a sip of water or even diet coke; however, please do report on eating or drinking that is part of your daily intake.” Prior EMA studies suggest that participants were able to reliably report the target behaviors of interest (56). Event-contingent recordings targeted information relevant to episodes of LOC eating. Consistent with much adult research regarding BE in the natural environment (57; 76; 95) adolescents were asked to make event-contingent reports before and after eating. Requiring participants to make before- and after-meal ratings (rather than after-meal ratings only) enabled examination of pre-meal predictors of LOC in the case that no random recording was logged before the episode.
- c. Interval-contingent. Interval-contingent responses occurred at pre-defined times of day, after school and before bed, to capture behaviors and variables that were not accounted for by other types of responses. An interval-contingent response scheduled at the end of the school day required participants to complete a

retrospective report immediately after school regarding the events and feelings that occurred during the school day, such that these events were captured retrospectively. Similarly, an interval-contingent response occurring immediately before bedtime enabled data to be gathered regarding interpersonal problems, negative affect or LOC eating episodes occurring after the final random prompt. Interval-contingent recordings assessed all constructs in the interpersonal model.

Through the combination of stratified random signal contingent recording and event recording, the strengths of each of these approaches were maximized. Additionally, using them in conjunction mitigated concerns about the inherent limitations of each approach. Notably, the use of random signals (particularly stratified random signals) allowed for accurate characterization of the daily experiences of participants. The addition of event-contingent recording ensured that we did not miss capturing data when target behaviors occurred. This combined approach offered a great deal of redundancy in the data collection.

### ***Measures captured***

- a. Positive and Negative Affect Schedule (PANAS) for Children (125). The PANAS is a 30-item self-report measure of positive and negative momentary affect. It has good psychometric properties, demonstrating excellent internal consistency (Cronbach's  $\alpha = 0.89 - 0.94$ ) and acceptable convergent and discriminant validity (125). Because the 30-item measure is overly burdensome for EMA use, we selected the six items which loaded highest on their respective factors (six for positive affect and six for negative affect). This approach is commonly accepted in EMA research (203). The PANAS for Children has been used extensively in EMA assessment as a brief,

reliable and valid means of measuring momentary emotional states (56; 188) and has been used in prior EMA studies among youth with LOC (94). In Elliott et al., 2009, however, the STAI-C (190), rather than the PANAS, was used to assess the ‘affect’ component of the interpersonal model. We selected to use the PANAS for a number of reasons. First, prior EMA studies of eating behavior among children (93) and adults with binge and bulimic behavior (187; 233) have used the PANAS as a measure of state affect, and therefore selection of the PANAS is consistent with prior EMA studies. Second, Elliott et al. used the trait-version of the STAI-C as a measure of anxiety over time, instructing youth to describe how they “usually feel.” Thus, although the interpersonal model was a good fit using the STAI-C, trait, as a measure of “affect,” the construct captured by the STAI-C, trait, did not fit the current objective of examining the interpersonal model in a momentary context. Finally, the STAI-C queries youth about symptoms and feelings of the affective state of “anxiety.” For example, questions include: “I worry too much,” and “My hands get sweaty.” The PANAS, by contrast, queries youth about a variety of distinct negative and positive emotion states. Thus, items included on the PANAS were determined to be more consistent with the construct of “negative affect,” and we therefore selected to use the PANAS rather than the STAI-C to operationalize the “affect” component of the interpersonal model.

- b. Eating Disorder Examination (61). LOC relevant items from the EDE were employed to assess LOC eating in an EMA compatible format. The specific items used to assess LOC eating included “Rate your *level of control* over eating,” “To what degree did you *lose control*...,” “Did you feel like your eating was out of

control...,” “Did you feel a sense of *loss of control*...,” and “Were you *able to stop eating*...” Using a similar format, LOC eating has been successfully assessed in past EMA protocols (56). Girls rated the degree to which LOC was experienced during the specified meal or snack using five-point Likert scales ranging from “No, not at all,” through “Yes, very much.” As a dichotomous variable, “high” LOC episodes included after-meal recordings in which adolescents reported a ‘4’ or higher (range 1 – 5, 1 = not at all, 5 = a lot/very much) on any LOC question comprising the LOC composite scale, which was derived by calculating a mean of relevant LOC items. All other episodes were categorized as “low” LOC.

- c. Social Adjustment Scale, Self-Report (235). Interpersonal problems were assessed using items from the ‘Interpersonal Problems’ subscale of the Social Adjustment Scale, Self-Report, which measures social functioning in four domains: school, friends, family, and dating. Items queried included the extent to which adolescents argued with someone, felt rejected, felt lonely, wished their relationships were better, and wished they had more friends. The construct of interpersonal problems has been successfully adapted for EMA assessment in past research (137).
- d. Posture. Posture significantly influences the HRV power spectrum, with a shift toward reduced HF power, and an increase in the LF:HF ratio, in standing, compared to supine positions (97; 160; 183). Studies examining differences in HRV as a function of supine versus other reclining postures (prone, left side, right side) suggest few differences in HRV indices (173; 232). With regard to a seated posture, one study found significant decreases in HF power, increased LF power in normalized units, and a greater LF:HF ratio, compared to prone position. A second study found

the expected stair-step pattern wherein inter beat interval, HF power, and respiratory sinus arrhythmia, all decreased significantly from supine to sitting to standing (232). Given significant variability of HRV across posture, all EMA recordings queried participants regarding their posture. Four options: reclining, sitting, and standing still, and walking, were presented.

Appendix 4 depicts EMA screens for all questions pertaining to interpersonal model variables.

### **Holter monitoring**

#### ***Data collection***

A 12-lead, 9 channel Mortara H12 Holter monitor system was used to measure HRV for two consecutive days. The Mortara H12 Holter monitor is compact and lightweight, weighing 4 ounces (129 g). Prior to applying electrodes, the skin was exfoliated, if necessary, and cleansed using rubbing alcohol, to maximize the electrical signal. After applying electrode leads, the signal quality was checked, and participants were familiarized with the technical equipment. During the Holter monitoring days, participants followed instructions not to bathe, shower, or engage in intense activity.

Data was sampled at a frequency of 1000 Hz, which falls above the range of 350-500 Hz recommended by the Task Force (216). The Holter monitor was synced in time with the EMA palm top computers to enable identification of Holter recording segments that correspond with self-reported variables. Generated ECG recordings were screened for clinically relevant abnormalities by an experienced cardiologist and were analyzed by the PI of the study, trained in use of the Holter equipment.

#### ***Data analysis***



Two primary types of analyses are used to analyze HRV, including time- and frequency-domain analyses.

Time-domain analyses are based on statistical computations of the variance in beat-to-beat intervals. The unit of measurement for HRV in time-domain analyses is milliseconds. Outcomes used in the current study include one measure of overall HRV, SDNN (the standard deviation of beat-to-beat intervals), and two measures of parasympathetically-mediated HRV, RMSSD (the root mean square of successive differences in beat-to-beat intervals), and pNN50 (the proportion of consecutive beat-to-beat intervals differing by greater than 50 ms). High variability in successive beat-to-beat intervals is indicative of a fast oscillatory frequency, implicating the parasympathetic nervous system; thus, RMSSD and pNN50, which reflect greater variability in successive beat-to-beat intervals, are thought to predominantly reflect parasympathetically-mediated HRV. Further, the Task Force (216) recommends that RMSSD, and pNN50 best represent HRV mediated by parasympathetic innervation (13).

Frequency-domain analyses is a second method by which HRV data can be analyzed, utilizing power spectral methods to partition the variance across beat-to-beat intervals based on frequency. Using this type of analysis, oscillatory components are extracted from the raw data (consecutive beat-to-beat intervals) using transformations such as the fast Fourier transformation or autoregressive modeling techniques. From oscillatory components, a power spectrum is generated which represents the distribution of variance occurring at difference frequencies (13). Oscillations occurring more rapidly (typically between 0.15 - 0.4 Hz) are considered high-frequency HRV (HF-HRV), whereas oscillations occurring more slowly (typically 0.04 - 0.15 Hz) are considered low-



frequency HRV (LF-HRV). Analyses of HRV data as an index of autonomic activity are grounded in the differences in oscillatory frequency produced by the parasympathetic, versus the sympathetic, systems. As noted above, HF-HRV is well accepted as a means of isolating parasympathetically-mediated HRV (158). By contrast, there is controversy surrounding respective parasympathetic and sympathetic contributions to LF-HRV, with LF-HRV thought to reflect actions of both systems.

In the current study, frequency-domain outcomes reported include HF-HRV (frequencies in the range of 0.15 - 0.4 Hz), LF-HRV (0.04 - 0.15 Hz), as well as the ratio between HF and LF HRV, thought to represent “sympathovagal balance” (216). Values can be expressed in either milliseconds squared or normalized units (216).

As recommended by the Task Force, frequency-domain analyses are appropriate for examining short-term variability in HRV, whereas time-domain methods are recommended for examination of HRV over longer intervals (e.g. 24-hour recordings). However, in ambulatory settings, RMSSD is also recommended as an index of vagal tone for reasons related to non-stationarity of data in ambulatory settings. Specifically, frequency-domain analyses applying Fourier transformation assumes that data show stationarity over time, meaning that the data generally have a stable mean and variance throughout the specified period. In ambulatory settings, this assumption is likely to be violated. The current study analyzed parasympathetically-mediated HRV in several ways including time-domain indices of RMSSD and pNN50 and frequency-domain indices of HF-HRV, LF-HRV and the ratio between HF- and LF-HRV.

To examine HRV as it relates to the components of the interpersonal model, the EMA monitoring equipment was synced in time with the Holter monitor. For random

recordings, segments of HRV data that occur immediately prior to the recording were identified. For eating-related recordings, segments of HRV data that occur 30 - 60 minutes prior to the recording were identified. Consistent with an existing study that concurrently employed self-report of mood and HRV monitoring in the natural environment (177), five-minute intervals immediately preceding self-report variables were selected for analysis. Five-minute segments are recommended by the Task Force for examining short-term fluctuations in HRV, although segments as short as one minute may be used (216).

### **Evaluation**

- a. Acceptability Questionnaire. A questionnaire assessing acceptability was administered to all participants following completion of the study. The questionnaire consists of five items rated on a seven-point Likert-type scale, as well as two open-ended questions regarding reasons for non-compliance and other comments. Questionnaire items pertain to how 'Easy,' 'Helpful,' 'Time-consuming,' and 'Disruptive' participants found the monitoring. Anchors are provided for Likert-type ratings of 1 (not at all) through 7 (extremely). Question five is a rating of how positive or negative (ranging from 1 = very negative to 7 = very positive) participants judged the overall study experience. Questions were scored according to Likert-type ratings and items for which greater scores indicate more negative experiences were reverse scored. A mean of the five items was generated to produce an overall acceptability rating.

## POWER ANALYSIS

The current study was designed to establish procedures, mitigate factors that could potentially interfere with effective testing of the interpersonal model according to the proposed procedures, and obtain variance estimates needed in determining the sample size for a formal, adequately powered study. Variance estimates will be obtained by calculating effect sizes, including  $R^2$  and pseudo  $R^2$  values. In ordinary least squares models,  $R^2$  values will be used to determine the amount of variability explained by the model and the  $\Delta R^2$  will be calculated to determine the amount of variability explained by variables of interest. For non-least squares models, as is the case for linear mixed models, pseudo  $R^2$  values will be calculated by subtracting the estimate of residual variance from the model including predictor variables from the estimate of residual variance from the null model. The difference in variance explained is the amount of variability explained by predictor variables, which constitutes the pseudo  $R^2$ . A pilot study with 30 degrees of freedom for error was considered adequate for obtaining a reasonably reliable estimate. We therefore recruited 30 adolescents for the current study.

## **CHAPTER 4: RESULTS**

### **DEMOGRAPHICS AND BASELINE MEASURES**

One hundred and seven adolescent girls were screened by telephone for participation in a research study on eating and emotions after responding to emails and direct mailings. Of those who did not meet initial screening criteria, the majority either did not report LOC eating (30%), were taking psychopharmacological medications impacting body weight (13%), or had a BMI < 85<sup>th</sup> percentile (8%). Nineteen percent of families did not return our phone calls. Of 53 (49.5%) adolescents who met initial inclusion criteria based on a telephone screening, 44 were interested in participating in the study and attended a baseline screening appointment. All participants signed informed consent prior to participation and were compensated for study participation.

During the screening visit, 33 out of 44 adolescents were determined to be eligible to participate in the study. Of the 11 girls excluded from the study during the screening visit, two met criteria for clinical depression; six adolescents did not experience LOC eating or experienced LOC eating less than twice per month; one adolescent refused to answer required interview questions about LOC eating and therefore study inclusion criteria could not be determined; and two adolescents experienced distress while completing study procedures, and they were therefore excluded on the grounds that the risks of participation were deemed to outweigh potential benefits in the opinion of the study investigators.

Three adolescents who met baseline screening criteria did not participate in the data collection phase of the study. One decided during the screening visit that she did not

want to participate in the study because her sister did not qualify for the study. Two adolescents dropped out of the study after the practice day, one due to scheduling difficulties, and the other did not provide a reason.

### **Demographics**

Adolescents who completed all study procedures did not differ on any demographic or anthropometric characteristics compared with the 14 adolescents who did not complete parts 2 and 3 of the study (Table 1). The final sample was comprised of 30 obese and overweight adolescents, the majority of whom were African American (63.3%), 26.7% were Caucasian, 3.3% Hispanic/Latino, and 6.7% biracial. Adolescents' BMI ranged from 24.94 – 54.59 (BMI ranging between 91<sup>st</sup> and 99<sup>th</sup> percentile) (120).

### **Loss of control eating**

At baseline, adolescents reported  $7.4 \pm 8.4$  LOC eating episodes in the month prior to assessment (range 2 – 39). About one-third (36.7%) of adolescents reported only subjective bulimic episodes, 30% reported only objective bulimic episodes, and the remainder reported both types of LOC episodes. The majority (19, 63.3%) of adolescents reported at least four LOC episodes within the month prior to assessment, and 56.7% of girls reported at least four episodes per month in the three months prior to assessment (Criteria A for BED as proposed in the DSM-5). Twenty-two (77.3%) girls endorsed associated features (e.g. eating rapidly, eating until uncomfortably full, and feelings of disgust, depression or guilt). Only seven adolescents (23.3%) reported marked distress surrounding the eating episode (Criteria C for BED). Utilizing a more lenient criterion to characterize distress surrounding eating episodes, 17 (56.7%) adolescents reported at least moderate distress. Based upon the DSM cutoff for Criteria C (report of “marked”

distress), three (10%) adolescents met criteria for BED, all of whom reported objective bulimic episodes. Based upon a more lenient cutoff for Criteria C (report of “moderate” distress), seven (23.3%) adolescents met criteria for sub-threshold BED, all of whom reported objective bulimic episodes.

### **Psychological measures**

#### ***Beck Depression Inventory (BDI-II)***

BDI-II scores ranged from 0 - 24 ( $M = 11.87$ ,  $SD = 6.08$ ), which falls into the mildly depressed range. Nearly half of girls ( $n = 13$ , 43.3%) reported BDI scores between 0 - 9, constituting minimal depressive symptoms. A third ( $n = 10$ , 33.3%) reported mild depressive symptoms (BDI score 10 - 16), and the remainder of girls (7, 23.3%) reported moderate depressive symptoms (BDI scores 17 - 24). Controlling for age, race, and BMI-Z score, BDI scores among participants with sub-threshold BED ( $M = 15.16$ ,  $SE = 2.50$ ) were non-significantly higher than among participants without sub-threshold BED ( $M = 10.87$ ,  $SE = 1.31$ ,  $p = 0.15$ , Figure 4).

#### ***State Trait Anxiety Inventory (STAI-C)***

Mean Total STAI-C anxiety score among adolescents in the study was 14.11 ( $SD = 7.5$ ,  $Range = 0 - 30$ ). Controlling for BMI-Z score and race, adolescents with sub-threshold BED, based on modified DSM-5 criteria described above, reported significantly higher anxiety ( $M = 19.80$ ,  $SE = 2.96$ ) compared to those in the non-BED group ( $M = 12.37$ ,  $SE = 1.60$ ,  $p = 0.04$ , Figure 4).

#### ***Kiddie Schedule for Affective Disorders and Schizophrenia (KSADS)***

The average value for total symptoms across all participants was 2.57 ( $SD = 3.17$ ,  $Range = 0 - 12$ ). A score of “2” is equivalent to one threshold-level psychiatric symptom,



or 2 sub-threshold symptoms, and a score of “3” is equivalent to one threshold-level psychiatric symptom and one sub-threshold symptom. The average value for “mood/internalizing” symptoms was 1.03 ( $SD = 1.30$ ,  $Range = 0 - 5$ ), and the average value for “behavioral/externalizing” symptoms was 1.50 ( $SD = 2.60$ ,  $Range = 0 - 10$ ).

Controlling for relevant covariates, no statistically significant differences were detected between adolescents with and without sub-threshold BED for total psychiatric symptoms ( $p = 0.25$ ). Adolescents without sub-threshold BED reported non-significantly more symptoms ( $M = 2.92$ ,  $SE = 0.66$ ) than those with sub-threshold BED ( $M = 1.41$ ,  $SE = 1.26$ ). For mood/internalizing symptoms, adolescents with ( $M = 1.07$ ,  $SE = 0.55$ ) and without ( $M = 1.02$ ,  $SE = 0.29$ ) sub-threshold BED reportedly equivalent symptom levels ( $p = 0.94$ ). For behavioral/externalizing symptoms, those without sub-threshold BED reported non-significantly more behavioral symptoms ( $M = 1.86$ ,  $SE = 0.54$ ) compared to those with BED ( $M = 0.33$ ,  $SE = 1.04$ ).

## **DATA CLEANING AND REDUCTION**

### **Outlier Screenings**

All data were screened for normality, and outliers were examined to determine plausibility. Logarithmic transformations were applied to variables violating normality assumptions, as described in respective sections. Across model aims, independent variables were not mean centered because no models contained simultaneous mean and interaction effects for which mean centering was needed in order to reduce multicollinearity. For non-EMA data, outliers were adjusted to fall 1.5 times the interquartile range below or above the 25<sup>th</sup> or 75<sup>th</sup> percentile, respectively (19). Outliers

were defined as values falling 1.5 times the interquartile range below or above the 25<sup>th</sup> and 75<sup>th</sup> percentile. In the majority of cases, findings from analyses in which outliers were converted were compared to those in which the raw data were retained. When possible, analyses retaining raw data are presented to preserve the integrity of the data (except in cases in which outliers were determined to be implausible).

## **EMA**

EMA data was examined for missing and incomplete recordings. Cases were considered incomplete if any question was unanswered. Incomplete cases (n = 34) were removed from the dataset. The dataset was examined for random recordings occurring within less than 10 minutes of previous random recording, and these cases (n = 13) were removed from the dataset.

Respective composite scores were calculated from five individual interpersonal items (e.g. “how much have you argued with someone?”), six positive affect items (e.g. “happy”), six negative affect items (e.g. “sad”), and four LOC eating items (e.g. “to what degree did you lose control during this eating episode?”). Cronbach’s alpha values were calculated to evaluate internal consistency (reliability) of each composite score. Cronbach’s alpha values for the interpersonal, positive affect, and negative affect composite scores were good (36), ranging between 0.89 and 0.93. Values, along with the individual items comprising each composite scale, are presented in Table 2. The Cronbach’s alpha value for the LOC eating composite derived from all four questions pertaining to LOC eating was 0.75, which falls into an acceptable range, however, this composite was reevaluated, excluding the item “Did you feel like you were able to stop eating during the meal you just finished?” When this item was excluded from the LOC

composite scale, the Cronbach's alpha value was improved to 0.89, and it was determined that the LOC construct was best represented by only three items.

High-threshold LOC episodes were categorized by identifying after-meal recordings in which adolescents indicated a '4' or higher (range 1 – 5, 1 = not at all, 5 = a lot/very much) on any LOC question comprising the LOC composite. This variable represented LOC dichotomously, intended to capture episodes in which a definite sense of LOC was experienced ("high-threshold LOC").

Aggregate levels of interpersonal problems, negative affect and positive affect, LOC and high-threshold LOC eating were derived by calculating the mean of all values reported on the respective variable over the course of the study period.

## **HRV**

HRV recordings were translated from proprietary format into binary code using the program SuperECG (Mortara Industries). A novel analysis program was applied to 24-hour Holter data using MATLAB (The Math Works, Natick, MA) software (courtesy of B. Fetters and R. Berger). The eight non-derived leads were viewed and the single lead in which noise was minimized was chosen. The program automatically excludes five-minute epochs if > 5% of beats are premature or noisy and one-hour segments if > 50% of five-minute epochs are rejected. Derivation of special periods (comprised of six-hour segments (00:00 – 5:59; 6:00 – 11:59; 12:00 – 17:59; 18:00 – 23:59)) required that all hours within that segment be present.

## PROTOCOL

Given the pilot status of the current study, several modifications were made to the EMA protocol at the onset of the study, in response to feedback from study participants. First, adolescents were not asked to complete an interval-contingent recording after school. While completion of such recordings was initially proposed in order to capture LOC episodes that occurred during school, it was determined that such reports would be retrospective in nature. Furthermore, such reports typically coincided with the completion of random recordings, beginning immediately following the school day (around 15:00 – 16:30). Therefore, the advantages of: (1) not over burdening adolescents by asking them to complete interval-contingent and event-contingent recordings that were temporally proximal in nature, and (2) excluding retrospectively-reported LOC episodes, were weighted greater than the potential advantages of capturing LOC episodes which occurred during school and other information reported during the interval-contingent report after school. Therefore, the decision was made to eliminate the after-school interval-contingent report from the EMA protocol.

Similarly, after several participants completed the study, it was determined that the event-contingent report occurring before bed would also be eliminated from the protocol. This change was made for several reasons. First, on many nights, the interval-contingent “before bed” report occurred at nearly the same time as the 21:50 random recording. Second, in the case that an adolescent experienced an eating episode after the final random recording, such episodes would, in theory, be captured by subsequent before- and after-meal event-contingent recordings. Therefore, in order to reduce burden by eliminating the redundancy of requiring a “before-bed” interval-contingent recording

proximal in time to the final (21:00) random recording, this element of the protocol was eliminated. Data from the first five participants' data revealed that adolescents completed fewer before- and after-meal ratings per day than expected ( $M = 1.41$  ( $SD = 0.78$ ) before-meal and  $M = 1.33$  ( $SD = 0.76$ ) after-meal ratings per day). The hypothesis that reducing burden may improve compliance with random and event-contingent recordings further contributed to the rationale for removing interval-contingent recordings from the EMA protocol. Given that the broad goal of the EMA protocol was to elucidate interpersonal and affective predictors of LOC eating, the before- and after-meal recordings were considered to be the most important recordings, and the importance of completing event-contingent recordings was emphasized to adolescents at baseline and subsequent visits.

## **HYPOTHESIS TESTING**

### **Aim 1**

To evaluate the feasibility and acceptability of using a naturalistic study design to test the interpersonal model of LOC eating among overweight adolescent girls who report LOC eating.

#### ***Hypothesis 1a.***

It was hypothesized that participants will be compliant with random recordings, defined as a response rate greater than or equal to 75%, and event-contingent recordings.

Twenty-nine participants were included in compliance analyses. One adolescent was excluded because she provided only four data points over approximately two hours. Two methods were employed to calculate compliance statistics. Compliance statistics

were calculated using data collected from all participants, such that adolescents who participated in the study for a longer duration had greater influence on overall compliance rates. Second, compliance was calculated for each adolescent, after which average rates of compliance were calculated, such that each adolescent equally influenced the overall compliance rate. Statistics for each method are presented in Table 3.

Twenty-nine adolescents completed 1069 random recordings over 387 days. Compliance with random recordings was calculated by dividing the number of random recordings completed within one hour of stratified anchor times (e.g. 11:10, 13:50, 16:30, 19:10, 21:50) by the total number of possible random recordings. Study compliance was 70.0%, which was lower than the expected compliance rate of 75%. Excluding adolescents with compliance rates lower than 50%, compliance was 74.1%. Compliance across participants ranged between 38% and 90% ( $M = 69.4$ ,  $SD = 13.2$ , Table 3).

Overall, adolescents completed 500 before-meal and 460 after-meal recordings. On average, adolescents completed 1.27 ( $SD = 0.69$ ) before-meal and 1.17 ( $SD = 0.62$ ) after-meal recordings per day. There was a wide range of compliance with before- and after-meal across participants. One subject only completed four event-contingent recordings throughout the entire duration (8 days) of participation in the study while other adolescents ( $n = 5$ ) completed two event-contingent recordings nearly every day of study participation.

At the conclusion of the study, adolescents were queried regarding common reasons that they were unable to complete recordings when asked. Adolescents were allowed to list as many reasons as they desired. Reasons provided by adolescents are depicted in Figure 6. The two most common reasons for missed ratings provided by



adolescents were participation in another activity such as sports practices or after school activities (cited eight times) and not having the device on hand (e.g. leaving the device in the car or at home, cited eight times). Additional common reasons included being busy (e.g. busy with family or friends, cited seven times), being asleep (cited six times) and forgetting (cited five times). Other infrequently cited reasons included being in a place where it was inappropriate to use the device (e.g. church, cited two times), doing chores (2), sick, tired, or “not feeling like it” (2), not being able to find the device (1), not paying attention (1), technical problems with the device (1), and being in the shower (1).

### ***Hypothesis 1b.***

Participants will attend all laboratory visits required for collection of EMA data.

Laboratory visit attendance was calculated to assess the feasibility of attending multiple visits required to conduct studies using EMA and Holter monitoring procedures. Non-baseline study visits were counted as either (a) attended, (b) canceled-rescheduled, (c) canceled-not rescheduled, or (d) no show. Lost data was tracked due to (a) missed visits and/or (b) equipment malfunction between visits. Procedures will be considered feasible if an attendance rate (including rescheduled visits) greater than 75% was achieved.

On average, adolescents completed 4.7 ( $SD = 0.65$ ,  $Range = 4 - 6$ ) study visits. Two adolescents who participated in the practice period did not wish to continue participating in the data collection phase of the study. No adolescent who participated in the data collection phase of the study was lost to follow-up during this phase. The vast majority of study visits were attended (103, 91.2%). Of those unattended, 8 (7.1%) were cancelled and rescheduled within 1 - 2 days of the original visit date. Seven of these

visits were rescheduled within 1 - 2 days, resulting in the completion of consecutive visits within seven days of each other, and one visit was rescheduled within four days, resulting in greater than seven days between consecutive visits. Two (1.8%) visits were not rescheduled, and in these cases adolescents returned on the date on which their subsequent visit was scheduled, resulting in greater than seven days between consecutive visits. The reason for visit cancellation was noted in all (3) cases in which a cancellation resulted in adolescents spending more than seven days between study visits. Reasons for cancelled visits included maternal chronic illness, being out of town, and schedule conflict due to the adolescent's other activities. Because the attendance rate exceeded 75%, the requirement that adolescents and their families attend multiple clinic visits for the purpose of EMA data collection was considered feasible.

Data loss due to equipment malfunction and other reasons was monitored throughout the duration of the study. Data was collected successfully on 363 days out of 429 days in which adolescents participated in the study. Rate of data loss was 15.4%. Reasons for data loss included programming error (49.15%), technical malfunction (e.g. broken screen, 18.64%), participant error (e.g. removing the batteries from palm top computer, 8.5%) or unknown reasons (11.9%). The bulk of lost data occurred at the onset of the study, while procedures were being implemented. Given that the current study was designed as a pilot study, a learning curve was expected. Excluding the first five participants, data was collected successfully on 324 out of 361 days, resulting in a 10.2% rate of data loss. Excluding these adolescents, reasons for data loss included technical malfunction (e.g. broken screen, 67.6%), participant error (e.g. removing the batteries from palm top computer, 13.5%) or unknown reasons (20.0%).

### ***Hypothesis 1c.***

The study procedures will be acceptable to overweight adolescent girls with LOC eating.

To assess acceptability, girls were administered an acceptability questionnaire (Appendix 4). Average rates of acceptability were calculated for each questionnaire item and for a total score. The procedure was considered 'acceptable' if a mean rating (across all questionnaire items) greater than or equal to '4' is achieved.

On a seven-point Likert-type scale, overall acceptability across participants was 5.30 ( $SD = 0.83$ ,  $Range = 3.00 - 7.00$ ). Despite that adolescents reported the self-monitoring to be moderately disruptive ( $M = 3.13$ ,  $SD = 1.81$ ) and time-consuming ( $M = 2.53$ ,  $SD = 1.41$ ), average ratings of self-monitoring on the dimension of "very negative" through "very positive" was 5.87 ( $SD = 1.07$ ,  $Range = 3.00 - 7.00$ ), reflecting generally positive perceptions of self-monitoring experiences. Since a mean acceptability rating (across all questionnaire items) of greater than '4' was achieved, the study procedures were considered to be 'acceptable.' Means, standard deviations, and ranges for overall acceptability and individual items are presented in Table 4.

### ***Hypothesis 1d.***

It was hypothesized that compliance rates, attendance and acceptability ratings would be unrelated to LOC eating, negative affect, interpersonal problems, age, race, and BMI. Independent variables for interpersonal problems, negative and positive affect, and LOC were derived by aggregating all reported values for each respective variable over the course of the study period.

Given that all adolescents completed between four and six study visits, and no adolescent was non-compliant with attendance requirements of the study, only the compliance and acceptability dimensions of feasibility were examined in relation to factors noted above: LOC eating, negative affect, interpersonal problems, age, race, and anthropometric variables.

Across dependent and independent variables, nine outliers were detected in five participants—five values for negative affect (log transformed), one for LOC eating, two for compliance, and one for height. All outlier values were plausible, and fell within four standard deviations of the mean. One subject's data included four outliers; consequently results were replicated excluding this subject from the analyses. Both sets of results are presented.

In the regression model predicting compliance by demographic and anthropometric factors ( $R^2 = 0.11$ ), age ( $p = 0.76$ ), race ( $p = 0.58$ ) height ( $p = 0.17$ ), and percent adiposity ( $p = 0.42$ ) were non-significant. Although demographic and anthropometric variables did not significantly contribute to the prediction of compliance, model fit was improved by the inclusion of these variables, and they were therefore included in subsequent models.

Due to the high degree of multicollinearity between interpersonal model variables (interpersonal problems, affect, LOC eating), each variable was examined independently. In the model predicting compliance by aggregate interpersonal problems ( $R^2 = 0.29$ ), there was a significant main effect of interpersonal problems ( $\beta = -0.46$ ,  $p = 0.02$ , Table 5), such that higher interpersonal problems were associated with lower compliance. Negative affect did not significantly contribute to the prediction of compliance ( $R^2 =$

0.24), however a non-significant pattern was revealed wherein greater aggregate negative affect over the study period was associated with lower compliance ( $\beta = -0.39, p = 0.06$ ). Aggregate positive affect was not associated with study compliance ( $R^2 = 0.12, p = 0.66$ ), controlling for the same covariates. In the model prediction of compliance by aggregate high-threshold LOC ( $R^2 = 0.42$ ), there was a significant effect of high-threshold LOC ( $\beta = -0.61, p < 0.01$ , Table 5), such that greater frequency of high-threshold LOC episodes was associated with lower compliance. Similarly, there was a marginal inverse association between LOC eating measured continuously and study compliance ( $R^2 = 0.23, \beta = -0.36, p = 0.07$ ). Therefore, in contrast to study hypotheses, compliance was related to two components of the interpersonal model: interpersonal problems and LOC eating.

Excluding the subject whose data contained outliers for several anthropometric and EMA variables, findings generally remained the same. Interpersonal problems ( $\beta = -0.41, p = 0.05, R^2 = 0.21$ ) and high-threshold LOC episodes ( $\beta = -0.50, p = 0.01, R^2 = 0.31$ ) were significantly related to study compliance. When the subject whose data comprised nearly 50% of outliers was excluded, the relationship between negative affect and compliance was tempered ( $p = 0.24$ ), as was the relationship between LOC eating ( $p = 0.26$ ) and compliance.

For acceptability, eight values were considered outliers across dependent and independent variables—five for negative affect (log transformed), two for high-threshold LOC eating and one for height. All outlier values were plausible. All values fell within four standard deviations of the mean (constrained by the scale ranges). One subject's aggregate EMA data consisted of a single LOC rating because she only completed one day of EMA data collection due to equipment malfunction. Hers, and one other

participant's, data comprised > 50% of outliers, so results were replicated excluding these participants from the analyses. Both sets of results are presented.

In the model of study acceptability ( $R^2 = 0.11$ ), demographic factors including age ( $p = 0.44$ ) and race ( $p = 0.30$ ) and anthropometric factors including height ( $p = 0.44$ ) and percent adiposity ( $p = 0.35$ ) did not contribute significantly, consistent with study hypotheses. However, model fit for subsequent models was improved by the inclusion of demographic and anthropometric variables, and these variables were therefore retained in subsequent models.

For LOC eating, high-threshold LOC was significantly related to lower acceptability ( $\beta = -0.52, p < 0.01, R^2 = 0.36$ ). As a continuous variable, this relationship did not reach significance but a similar pattern was observed ( $\beta = -0.28, p = 0.15, R^2 = 0.19$ ). There was also a marginal relationship between negative affect and acceptability such that greater negative affect was non-significantly related to lower acceptability ratings ( $\beta = -0.37, p = 0.07, R^2 = 0.24$ ). Interpersonal problems ( $p = 0.27$ ) and positive affect ( $p = 0.52$ ) were found to be unrelated to study acceptability.

When outliers were excluded, relationships between interpersonal problems ( $p = 0.57$ ), negative ( $p = 0.09$ ) and positive ( $p = 0.61$ ) affect and acceptability remained the same. For LOC eating, relationships with study acceptability were no longer observed. Neither high-threshold LOC ( $p = 0.20$ ) nor LOC ( $p = 0.73$ ) was significantly related to acceptability, suggesting that this association was driven by outlier values.



## **Aim 2**

To evaluate the concordance of self-reported negative affect and autonomic indices (heart rate (HR) and heart rate variability (HRV)) among overweight adolescent girls with LOC eating.

### ***Hypothesis 2.***

Self-reported negative affect will be correlated with the following time- and frequency- domain indices of HRV in the following directions: Positive associations are expected to be found with respect to HR; inverse associations are expected to be found with respect to parasympathetically-mediated indices of HRV; the association between overall HRV and negative affect is an exploratory aim.

The concordance of HR and HRV and self-reported negative affect was examined at three temporal levels. First, the association between 24-hour HR and HRV indices and trait negative affect, measured using trait measures of depression and anxiety symptoms, was examined. Second, the association between 24-hour HR and HRV indices and aggregate daily state affect was examined. Finally, the associations between momentary physiologic indices and momentary state negative affect were examined. Indices of HR and HRV varied depending on the temporal level of the analyses. Twenty-four hour HR and HRV indices included beats-per-minute (heart rate (HR)), SDNN, and HF power (normalized units). Momentary HR and HRV indices included beats-per-minute (HR), SDNN, RMSSD, pNN50, and HF-HRV.

Thirty adolescents completed 24-hour HRV recordings over 60 days. However, thirteen (21.7%) recordings were excluded because the data was not saved in the proper format; seven (11.7%) electrocardiograms were completed on data cards that did not

include a “high-definition” feature, which disabled the study team from processing the data using the proposed methodology; two (3.3%) data cards were returned without electrocardiography data; and one (1.7%) adolescent reported that the adhesives did not remain in place. The majority of the errors noted above were corrected after the first several participants completed the study, and, given the pilot nature of the study, a learning curve was expected. The hypothesized reason why data cards were returned without electrocardiography data is the following: Because data cards recorded up to 24-hours of ECG data, adolescents were asked to change the data card between days 1 and 2 of the Holter monitoring procedure. Although all adolescents were instructed regarding how to initiate the ECG recording, initiated the first ECG recording with a study team member present, and were provided type-written instructions regarding this procedure, it is thought that two adolescents did not accurately initiate the recording on day 2. Therefore, data from 19 participants was available for analysis and aggregate 24-hour HR and HRV data is presented in Table 6.

#### *24-hour HRV and trait negative affect*

Indices of trait negative affect included: (1) depressive symptoms as measured by the total score on the Beck Depression Inventory (BDI); (2) anxiety symptoms as measured by the total score on the State Trait Anxiety Inventory- Child (STAI-C); (3) total psychiatric symptoms as reported on the Kiddie Schedule for Affective Disorders and Schizophrenia –Screen version (KSADS) interview; (4) “mood/internalizing” symptoms (consisting of symptoms of depression, suicidal ideation, mania, and panic disorder) reported on the KSADS; and (5) “behavioral/externalizing” symptoms (consisting of symptoms of attention deficit hyperactivity disorder, oppositional defiant

disorder, conduct disorder, and alcohol and drug use) reported on the KSADS. Symptoms were categorized as absent (0), sub-threshold (1) or present (2), and scores were derived by summing items comprising respective scales.

Across dependent and independent variables, 13 outliers were detected, distributed across five participants. Three outliers were detected for height, seven for KSADS symptoms, and three for HR and HRV variables. All outliers were plausible, falling within 3 - 4 standard deviations of mean values. Two participants' data comprised > 50% of outlier values. One of these participants was prescribed a psychoactive antidepressant (Sertraline). Despite literature suggesting that selective serotonin reuptake inhibitor medications do not significantly alter HRV indices (108) (providing a rationale for case-by-case inclusion of participants taking such medications), this participant was excluded from the analyses given her medication status and the fact that her data constituted outlier values on several primary dependent and independent variables. This participant's data was excluded from all subsequent HRV analyses in order to maintain homogeneity for medication status. Other outlier values were retained in analyses and did not significantly alter the pattern of findings.

Excluding this participant, 33 days, for 18 participants, were included in analyses examining relationships between trait negative affect (e. g. depression, anxiety) and physiologic indices. In the mixed model prediction of heart rate, as expected, there was a non-significant trend for older age to be inversely associated with heart rate (estimate = - 2.40,  $p = 0.06$ ), such that as age increased, heart rate decreased. No other demographic variable including race ( $p = 0.90$ ) or anthropometric variable including height ( $p = 0.58$ ) or percent adiposity ( $p = 0.36$ ) was significantly associated with heart rate. In the model

examining heart rate, there was a marginal, non-significant relationship such that greater trait anxiety symptoms were associated with higher heart rate (estimate = 0.47,  $p = 0.11$ ), controlling for covariates ( $R^2 = .01$ ,  $\Delta R^2 < 0.01$ ).  $\Delta R^2$  below .01 suggested that baseline anxiety symptoms explained <1% of variability in daily heart rate. No significant effects were observed for trait depressive symptoms ( $p = 0.89$ ), total psychiatric symptoms ( $p = 0.56$ ), mood/internalizing symptoms ( $p = 0.33$ ), or behavioral/externalizing symptoms ( $p = 0.87$ ). No demographic or anthropometric covariate significantly contributed to SDNN (all  $p$ 's  $> 0.05$ ), but model fit was improved by the inclusion of these variables in the model. For SDNN, no significant effects were observed for trait anxiety symptoms (estimate = - 0.79,  $p = 0.23$ ), trait depressive symptoms ( $p = 0.95$ ), total psychiatric symptoms ( $p = 0.90$ ), mood/internalizing symptoms ( $p = 0.67$ ), or behavioral/externalizing symptoms ( $p = 0.99$ ), controlling for demographic and anthropometric covariates.

In the mixed model prediction of normalized HF power, no covariate significantly contributed to the model, yet they were included in subsequent models because model fit was improved. Although significant findings were not observed for the relationships between total trait psychiatric symptoms and HF power or mood/internalizing symptoms and HF power, relationships were in the expected direction such that greater total symptoms (estimate = - 0.01,  $p = 0.12$ ,  $R^2 = 0$ ) and internalizing symptoms (estimate = - 0.04,  $p = 0.09$ ,  $R^2 < 0.01$ ) were associated with lower HF power (Figure 7). Trait depressive symptoms ( $p = 0.53$ ), anxiety symptoms ( $p = 0.49$ ), and behavioral/externalizing symptoms ( $p = 0.49$ ) were unassociated with normalized HF power.

### *24-hour HRV and daily affect*

Indices of daily affect included positive and negative affect and boredom. For each state affect variable, mean, maximum, and standard deviation, representing variability over the course of the day, were examined in relation to HR and HRV indices.

Thirty days of HRV recordings were completed on days on which EMA data was collected. Therefore, 30 days, from 17 participants, were included in analyses. Four outliers were detected across three participants: two for the standard deviation of positive affect, one for the standard deviation of interpersonal problems, and one for height. All outliers were plausible. Findings remained unchanged when outliers were converted, and therefore analyses containing original data are presented. Negative affect was log transformed to address skew.

Adolescents meeting sub-threshold criteria for BED ( $n = 7$ ) reported higher aggregate daily LOC eating ( $M = 2.33$ ,  $SD = 0.47$ ,  $p = 0.01$ ) compared to those not meeting sub-threshold BED criteria ( $n = 22$ ,  $M = 1.72$ ,  $SD = 0.54$ ). Groups did not differ on any other interpersonal model variable. Aggregate daily means and standard deviations for interpersonal problems, positive and negative affect, and LOC eating are summarized in Table 7.

Covariates considered in models examining HR included race, height, and percent adiposity. In models examining 24-hour HR in relation to indices of daily state affect, no significant effects were observed for mean daily negative (log transformed,  $p = 0.84$ ) or positive ( $p = 0.79$ ) affect, or boredom ( $p = 0.49$ ). The same pattern of findings was noted for maximum daily level of negative and positive affect and boredom (all  $p$ 's  $> 0.05$ ). In models examining SDNN, controlling for race, height, and percent adiposity, mean daily



negative affect (log transformed,  $p = 0.79$ ), positive affect ( $p = 0.66$ ), and boredom ( $p = 0.33$ ) were not significant. Results were the same when maximum values were examined.

For the examination of normalized HF power, after controlling for height and percent adiposity, mean daily negative affect was significant in the model such that greater daily negative affect was associated with lower HF normalized power (estimate =  $-0.17$ ,  $p = 0.04$ ,  $R^2 = 0.08$ ,  $\Delta R^2 = 0.08$ , Figure 8). Similar patterns were found for the maximum daily value of negative affect (estimate =  $-0.02$ ,  $p = 0.08$ ,  $R^2 = 0$ ) and the standard deviation (variability) of daily negative affect (estimate =  $-0.08$ ,  $p = 0.04$ ,  $R^2 = 0$ ). By contrast, neither mean daily positive affect (estimate =  $0.01$ ,  $p = 0.25$ ), nor mean daily boredom (estimate =  $0.01$ ,  $p = 0.26$ ) significantly contributed to the prediction of HF normalized power (Figure 8).

#### *Momentary HRV and state negative affect*

The same indices of state affect that were examined on a daily level were examined momentarily. These included momentary negative affect, positive affect, and boredom. Similar to prior analyses, momentary physiologic measures included HR, SDNN, and normalized HF power. In this case, normalized HF power was operationalized using the total HF power divided by the total power (HF power plus LF power (HF power / [HF power + LF power])). Two additional indices of parasympathetically-mediated HRV, RMSSD and pNN50, were also examined at the momentary level.

A number of covariates were considered, given their known correlations with HR and the respiratory sinus arrhythmia. First, adolescents categorized their posture and



activity level as either “reclining,” “sitting,” “standing still” or “walking.” Findings from mixed model regressions of posture on HRV indices, as well as existing studies suggesting that the most prominent differences in HRV exist between supine versus upright postures (97; 155), suggest that posture may be categorized as supine versus upright (“reclining” versus the other three categories (“sitting,” “standing still,” and “walking”)). Consistent with a prior study examining momentary HRV, time of day was not examined as a covariate (177), despite temporal variation in HR. Furthermore, the majority of recordings completed by adolescents occurred in the afternoon and evening, suggesting less variability in HR attributable to time of day, as compared to the amount of variability that would occur if recordings were completed randomly throughout a 24-hour period. Finally, the impact of type of recording (random, before-meal, after-meal) was considered in all models, given the known impact of eating behavior on HR and HRV. Therefore, the association between momentary state affect and HR and HRV indices was investigated in three models. First, the impact of affect was examined across all types of EMA recordings completed by adolescents. Next, the impact of state affect was examined separately for each type of recording. Finally, an interaction model was examined wherein the main effects of state affect and type of recording, and the interaction between state affect and type of recording, were tested.

One hundred and seventy EMA recordings, from 18 participants, were concurrent with HRV data collection. Data from the participant who endorsed psychotropic medication use was excluded. In addition, HRV segments with > 35% missing or noisy data were excluded, resulting in 128 cases of matched EMA to HRV data from 17 participants. Eight outliers were detected for HR and HRV variables. EMA data

contained outliers for the negative affect variable only, and this variable was transformed to address skew. All outliers were plausible. Findings remained unchanged when outliers were converted, and therefore analyses containing original data are presented.

The most prominent effects of momentary affect on HR indices were seen for the effects of negative affect on HR. Controlling for posture, there was a main effect for negative affect (log transformed, estimate = 22.21,  $p = 0.001$ ,  $R^2 = .18$ ,  $\Delta R^2 = 0.08$ ).  $\Delta R^2$  when negative affect is added to the model suggests that 8% of variance in momentary heart rate is attributable to self-report momentary negative affect. Split file analyses suggested that the relationship between negative affect and HR was strongest at random times (estimate = 21.01,  $p = 0.02$ ,  $R^2 = .17$ ,  $\Delta R^2 = 0.03$ ) and before meals (estimate = 29.12,  $p = 0.04$ ,  $R^2 = .19$ ,  $\Delta R^2 = 0.12$ ), with significant effects observed at each of these times, but not after meals ( $p = 0.45$ , Figure 9). The model fit was improved by the inclusion of rating type, yet no main effect of rating type ( $p = 0.11$ ) or interaction between rating type and negative affect ( $p = 0.55$ ) on HR was observed, even though the main effect for negative affect persisted (estimate = 13.14,  $p < 0.01$ ,  $R^2 = 0.24$ ,  $\Delta R^2 = 0.05$ ).

No effects of momentary negative affect were seen on overall HRV (SDNN, all  $p$ 's  $> 0.63$ ) or parasympathetically-mediated indices of HRV (RMSSD, pNN50, and HF/HF+LF, all  $p$ 's  $> 0.12$ ). However, the relationship between momentary negative affect and pNN50 was in the expected direction such that instances of higher reported negative affect were associated with lower parasympathetically-mediated HRV as measured by pNN50 (estimate = - 6.67,  $p = 0.12$ ,  $R^2 = 0.09$ ,  $\Delta R^2 = 0.02$ ). Table 8 presents estimates of fixed effects and significance values for three model types: models

including all types of recordings, split file models in which random, before-meal, and after-meal recordings were examined separately, and interaction models wherein the main effects of state affect and type of recording, and the interaction between state affect and type of recording, were tested jointly.

For positive affect, the association between positive affect and HR was in the expected direction (estimate = - 1.50,  $p = 0.14$ ,  $R^2 = 0.14$ ,  $\Delta R^2 = 0.03$ ), but was not significant. Nor was there a significant interaction between positive affect and rating type ( $p = 0.19$ ). No relationships were found between positive affect and the majority of HRV indices including models of SDNN ( $p$ 's for main effect, split file, and interaction models  $> 0.57$ ), RMSSD ( $p$ 's for main effect, split file, and interaction models  $> 0.39$ ), and pNN50 ( $p$ 's for main effect, split file, and interaction models  $> 0.35$ ). In the interaction model for HF/HF+LF, a marginally significant effect was observed such that rating type ( $p = 0.08$ ) and the interaction between rating type and positive affect ( $p = 0.08$ ) were marginally significant predictors of HF/HF+LF ( $R^2 = 0.05$ ,  $\Delta R^2 = 0.04$ ). Specifically, the relationship between positive affect and HF/HF+LF at random times (relative estimate = 0.06,  $p = 0.04$ ) differed significantly from that between positive affect and HF/HF+LF after meals (Figure 10).

Similar to positive affect, no associations were revealed between momentary boredom and HR and HRV indices, including for heart rate ( $p$ 's for main effect, split file, and interaction models  $> 0.23$ ), SDNN ( $p$ 's for main effect, split file, and interaction models  $> 0.21$ ), RMSSD ( $p$ 's for main effect, split file, and interaction models  $> 0.45$ ), pNN50 ( $p$ 's for main effect, split file, and interaction models  $> 0.41$ ), and HF/HF+LF ( $p$ 's for main effect, split file, and interaction models  $> 0.17$ ).

### **Aim 3**

To evaluate the validity of the interpersonal model positing the mediation hypothesis that interpersonal problems precede and predict negative affect, negative affect precedes and predicts LOC eating, and negative affect mediates the relationship between interpersonal problems and LOC eating.

Steps of the model involving negative affect were tested using both self-report of negative affect and HR and HRV indices as a physiologic proxy for negative affect. Lacking strong concordance,, self-report and physiologic variables were not combined into a latent construct.

#### ***Self-report variables***

Examination of links between self-reported components of the interpersonal model were examined in two ways. The first method used all available data to test relationships between model components and the second method utilized only data included in the mediation model. In order to be included in the mediation model, data must comprise one of the following three consecutive EMA recordings completed by the same participant during the same day: (1) a random recording, providing information regarding interpersonal problems (time 1), (2) a random or before-meal recording, providing information regarding negative affect (time 2), or (3) an after-meal recording, providing information about LOC eating (time 3). One-hundred and eighty four such cases were present. This set of data was used to test each path of the model within a complete mediation model. However, inclusion of all available data engendered greater

power for tests of individual paths between model components. Both sets of analyses are presented.

### ***Physiologic variables***

Given the non-stationarity of ambulatory HRV indices, it was determined that a procedure wherein a greater number of HRV data points (rather than a single HRV data point) would produce a more reliable test of the relationship between components of the interpersonal model. In mathematics, stationarity of data refers to the stability of statistical parameters (mean, variance) when shifted in time or space. Since HRV is subject to biological oscillations, it is considered non-stationary. Thus, incorporation of multiple measurements may be used to minimize error imposed by the analysis of very short HRV segments as described by the Task Force (216).

Two strategies were considered for improving stationarity. The first potential strategy considered was calculating the average of consecutive five-minute HRV segments throughout a pre-determined time preceding eating episodes. A second potential strategy considered was calculating the trajectory of five-minute HRV segments throughout a pre-determined period of time preceding eating episodes. The latter strategy was chosen for the following statistical and theoretical rationale. First, examination of trajectories instead of average values maximized the proportion of data included in analyses, which improved the statistical power of the test (considered a key factor in light of the limited number of eating episodes reported on days on which participants engaged in HRV monitoring). From a theoretical perspective, the latter strategy preserved the raw data from each five-minute segment rather than reducing it to an average value, and thereby provided more detailed information about participants' affective experiences



preceding eating episodes. Therefore, trajectories of each dimension of HR and HRV using linear, quadratic, and cubic functions throughout the time preceding eating episodes were modeled. The rationale for inclusion of quadratic and cubic functions was to examine potential non-linear associations between variables of interest. In this case, inclusion of a cubic function allows the examination of the hypothesis that the relationship between HR/HRV and eating episode type over time is characterized by two inflection points, typical of phasic reactivity and recovery. In the case that these more complex (quadratic and cubic) were insignificant, they would be dropped from models to improve parsimony. In such multilevel models, the linear term (time) denotes the rate of change of the HR or HRV variable; the quadratic term ( $\text{time}^2$ ) denotes the acceleration in rate of change; and the cubic term ( $\text{time}^3$ ) denotes the change in acceleration of rate of change. When more than one eating episode was reported in a single day, the time between eating episodes was calculated. If episodes were separated by more than three hours, both episodes were included in analyses. Inclusion of multiple episodes within a single day may confound the relationships between HRV fluctuations that are consequent to eating versus those that are interpersonal or psychological (affective) in nature. However, given the pilot nature of the study, inclusion of multiple episodes per day can be used as a strategy to maximize power.

The hypotheses involving physiologic measures of affect include hypothesis 3b, the link between interpersonal problems and affect, and 3c, the link between affect and LOC eating. Given the choice to examine trajectories rather than HRV at a discrete time point, the mediation model could not be examined using a physiologic index of affect.



### *Hypothesis 3a.*

Interpersonal problems will precede and predict LOC eating episodes (path c).

#### *Self-report*

Adolescents reported interpersonal problems during random and before-meal EMA recordings and they reported degree of LOC eating at after-meal recordings. Four-hundred and twenty-six after-meal recordings were preceded by a random or before-meal recording. The average interval between measurement of interpersonal problems and state affect was 28:37.

In the mixed model of LOC eating, interpersonal problems were significantly positively associated with LOC eating (estimate = 0.18,  $p < 0.01$ ,  $R^2 < 0.01$ ) such that greater interpersonal problems at time 1 predicted greater subsequent LOC eating (Figure 11).

In the mediation model, interpersonal problems and LOC eating were not measured at consecutive EMA recordings, but rather are parted by an “affect” measurement, occurring between the measurement of interpersonal problems and LOC eating. One-hundred and eighty four cases included three consecutive EMA recordings. Such constellations of EMA data within a single participant, over a single day, comprised the data used to analyze the mediation model.

Examining the impact of interpersonal problems (time 1) on LOC eating (time 3), path c, interpersonal problems predicted LOC eating (estimate = 0.31,  $p < 0.01$ ,  $R^2 = 0.05$ ). Examining the impact of both baseline interpersonal problems (time 1) and the change in interpersonal problems (time 2) within a single model, baseline interpersonal

problems (estimate = 0.32,  $p = 0.01$ ), but not the change in interpersonal problems ( $p = 0.93$ ), significantly predicted LOC eating ( $R^2 = 0.04$ ).

### ***Hypothesis 3b.***

Interpersonal problems will precede and predict negative affect (path a).

### ***Self-report***

Adolescents reported interpersonal problems at random times and before meals, and they reported state affect, including negative affect, positive affect, and boredom, during all types of EMA recordings. In order to control for the impact of eating on subsequent mood, variables measured before and after eating were excluded at time 1, and variables measured after eating were excluded at time 2. Negative affect was log transformed to address skew.

Six-hundred and forty-one non-meal recordings were completed prior to a subsequent random recording or before-meal recording. The average interval between measurement of interpersonal problems and state affect was 2:00:17. Controlling for negative affect at time 1, interpersonal problems at time 1 did not predict subsequent (time 2) negative affect (estimate = 0.01,  $p = 0.11$ ,  $R^2 = 0.15$ ), however the relationship between interpersonal problems and negative affect was in the expected direction, such that greater interpersonal problems were non-significantly associated with greater negative affect (Figure 12). Although the effect size for the overall model is medium,  $\Delta R^2$  for interpersonal problems was 0, suggesting that interpersonal problems did not contribute to the explained variance.

There was no relationship between interpersonal problems at time 1 and positive affect at time 2 ( $p = 0.45$ ), controlling for baseline positive affect. By contrast,

interpersonal problems at time 1 predicted greater levels of boredom at time 2 (estimate = 0.25,  $p = 0.004$ ), controlling for baseline level of boredom ( $R^2 = 0.07$ ,  $\Delta R^2$  for interpersonal problems < 0.01, Figure 12).

Examining the 184 cases including all interpersonal model variables, interpersonal problems predicted negative affect, controlling for baseline negative affect (estimate = 0.03,  $p = 0.04$ ,  $R^2 = 0.27$ ,  $\Delta R^2$  for interpersonal problems = 0.01). Interpersonal problems did not predict subsequent boredom ( $p = 0.89$ ), or positive affect ( $p = 0.77$ ), controlling for respective baseline affective states.

### *Physiologic*

Excluding ratings completed by the participant taking an SSRI, 93 reports of interpersonal problems were recorded on days on which HRV was examined. Thirty recordings were followed by a before- or after-meal recording occurring within 3 hours of the interpersonal problem recording, considered to denote that eating, a potential confounder of cardiovascular indices, had occurred. These recordings were therefore excluded from analyses. Fifteen additional episodes were preceded by a report of interpersonal problems within the previous 3 hours. To avoid confounding, all recordings occurring less than 3 hours following a prior recording were excluded from analyses. Therefore, 48 episodes, reported over the course of 29 days by 14 participants, were included in final analyses.

Interpersonal problems were categorized as “high” or “low” based on a cutoff of two on the interpersonal problems composite scale. Based on this cutoff, 13 recordings were categorized as “high” interpersonal problems, and the remaining 35 were categorized as “low” interpersonal problems.

Models examined the impact of interpersonal event on heart rate, SDNN, RMSSD, pNN50, and HF/HF+LF. Trajectories of the linear, quadratic, and cubic effects of time, in addition to the interaction effects of interpersonal event with each time function, constituted the independent variables. Table 9 presents model coefficients and significance values for each model.

For HR, there was a significant main effect in the positive direction for interpersonal problems (estimate = 13.78,  $p < 0.001$ ), suggesting that at the time that interpersonal problem ratings were completed, higher interpersonal problems were associated with elevations in HR. Specifically, a “high” interpersonal problem rating was associated with an average 14 beat increase in HR. By contrast, there were no significant main effects of any time functions (all  $p$ 's  $> 0.07$ ), meaning that HR did not change significantly following completion of interpersonal problem ratings, nor were there significant interactions between interpersonal problems and any time function (all  $p$ 's  $> 0.57$ ), suggesting that the trajectories of HR did not differ according to whether high versus low levels of interpersonal problems were experienced.  $R^2 = 0.05$  and  $\Delta R^2$  for interpersonal problems and interpersonal problem interactions = 0.03.

Similar to HR, two indices of HRV evidenced significant main effects for interpersonal problems. For RMSSD and pNN50, inverse relationships were revealed wherein higher interpersonal problems were associated with lower RMSSD (estimate = -14.74,  $p = 0.04$ ) and pNN50 (estimate = -6.33,  $p = 0.001$ ). Linear, quadratic, and cubic effects of time were not significant ( $p$ 's  $> 0.12$ ), suggesting no main effects of time on HRV following interpersonal problem ratings. Similarly, there were no significant interactions between interpersonal problems and any time function, indicating that the

trajectories of RMSSD and pNN50 did not differ following high versus low interpersonal problem ratings.  $R^2$  for RMSSD  $< 0.01$  and for pNN50  $= 0.02$ .  $\Delta R^2$  for interpersonal problems and interpersonal problem interactions for RMSSD  $< 0.01$  and for pNN50  $= 0.02$ . For HF/HF+LF, however, a different pattern of findings was noted wherein there was no main effect for interpersonal problems ( $p = 0.73$ ), meaning that high-frequency power was no different at the time of high versus low interpersonal problem ratings. Yet, significant main effects were noted for linear, quadratic and cubic functions of time (*all*  $p$ 's  $< 0.01$ ), and significant interactions were revealed between interpersonal problems and all time functions (*all*  $p$ 's  $< 0.05$ ). Specifically, there was a significant difference in the rate of change (slope) of HF/HF+LF following high, as compared to low, interpersonal problem ratings, as evidenced by the significant estimate for the linear function of time by interpersonal problem interaction term (estimate  $= -0.28$ ,  $p < 0.01$ ), a significant difference in the acceleration (or deceleration) in rate of change (estimate  $= 0.18$ ,  $p = 0.02$ ), evidence by the significant interaction effect of the quadratic function of time and interpersonal problems, and finally, a difference in the change in acceleration in rate of change (estimate  $= -0.03$ ,  $p = 0.05$ ), as evidenced by the significant interaction between the cubic function of time and interpersonal problems ( $R^2 = 0.05$ ).

Finally, for SDNN, there was no main effect for interpersonal problems, suggesting no baseline difference in SDNN between high and low interpersonal problems. Main effects for linear (estimate  $= -28.44$ ,  $p < 0.01$ ) and quadratic (estimate  $= 15.73$ ,  $p = 0.05$ ) functions of time were revealed, suggesting a significant decrease, followed by a stabilization, in the SDNN trajectory in the three hour time frame following all interpersonal problem ratings, regardless of whether interpersonal problems

were rated as high versus low. Absence of significant interactions between any time function and interpersonal problems (*all p*'s > 0.28) revealed that there were no differences in SDNN trajectories based on interpersonal problem level. Overall model  $R^2 = 0.02$  and  $\Delta R^2$  for interpersonal problems and interpersonal problem interactions < 0.01.

### ***Hypothesis 3c.***

Negative affect will precede and predict episodes of LOC eating (path b).

### ***Self-report***

For this hypothesis, two models: (1) the impact of state affect (time 1) and (2) the joint impact of state affect (time 1) and change in state affect (time 2) on LOC eating (time 3) were examined. Adolescents reported state affect at random recordings and before meals, and they reported LOC eating after meals. In order to control for the impact of eating on subsequent mood, variables measured before and after eating were excluded at time 1, and variables measured after eating were excluded at time 2.

Four-hundred twenty-six after-meal recordings were preceded by a random or pre-meal affect report. In the first model prediction of LOC eating based on negative affect, negative affect significantly contributed to the prediction of LOC eating (estimate = 1.37,  $p = 0.05$ ,  $R^2 = 0$ , Figure 13). By contrast, neither positive affect ( $p = 0.86$ ) nor boredom ( $p = 0.37$ ) significantly predicted LOC.

In the second model examining the impact of baseline negative affect (time 1) and change in negative affect (time 2), only higher baseline negative affect (estimate = 1.29,  $p = 0.04$ ,  $R^2 = 0.01$ ,  $\Delta R^2 = 0$ ) predicted LOC eating. The relationship between change in negative affect and LOC eating was non-significant, and opposite the direction expected (estimate = - 0.92,  $p = 0.13$ ,  $R^2 = 0.01$ ,  $\Delta R^2 = 0$ , Figure 13).



For positive affect, neither baseline level ( $p = 0.71$ ) nor change ( $p = 0.88$ ) in positive affect predicted LOC eating. For boredom, baseline (time 1) level of boredom (estimate = 0.13,  $p = 0.03$ ,  $R^2 = 0.01$ ,  $\Delta R^2 = 0$ ) was positively associated with LOC eating, but change in boredom (time 2,  $p = 0.35$ ) was not ( $R^2 = 0.01$ ,  $\Delta R^2 < 0.01$ ).

### *Physiologic*

Forty-nine eating episodes were reported by 17 participants on days on which HRV was examined. Four eating episodes occurring within three hours of a previous episode were excluded from analyses, as were two episodes reported by the participant who was taking an antidepressant. Therefore, 43 episodes, reported over the course of 27 days by 17 participants, were included in final analyses. Seven episodes were categorized as LOC, based on the previously defined criterion for “high-threshold LOC” by indication of a ‘4’ or higher (range 1 – 5, 1 = not at all, 5 = a lot/very much) on any LOC question comprising the LOC composite scale. The remaining episodes were categorized as non-LOC. Analyses were replicated using a more lenient criterion for LOC (values exceeding two on the LOC composite scale). In this set of analyses, 14 episodes were categorized as LOC and results were the same.

HR and HRV trajectories within 3 hours preceding the after-meal recording, excluding the half hour immediately prior to the recording, constituted the dependent variable. The rationale for excluding the half hour immediately preceding the after-meal recording was due to the known impact of food intake on HRV (70; 129). On average, before-meal recordings were completed 28:37 minutes prior to after-meal recordings. Therefore, it was assumed that the half hour immediately preceding the after-meal recording was spent eating.

Models examined the impact of eating episode type on HR, SDNN, RMSSD, pNN50, and proportion of HF power divided by total power. Trajectories of the linear, quadratic, and cubic effects of time, in addition to the interaction effects of LOC with each time function, constituted the independent variables. Table 10 presents model coefficients and significance values for each model.

For HR, there was a significant main effect of LOC, suggesting that at the time immediately preceding the eating episode, HR was significantly higher prior to LOC, as compared to non-LOC episodes (estimate = 38.49,  $p = 0.04$ ). Linear, quadratic, and cubic effects of time were not significant ( $p$ 's > 0.12), suggesting no main effects of time on HR prior to eating. However, interaction effects between episode type and quadratic and cubic functions of time suggested that the trajectories of heart rate differed significantly by eating episode type. Specifically, the significant, positive estimate of the cubic function of time by LOC interaction term (estimate = 11.52,  $p = 0.02$ ) suggests a greater initial increase in heart rate preceding LOC episodes compared to non-LOC episodes, depicted by the steep rise in the curve representing HR prior to LOC episodes in the approximate 2.25 – 3 hours preceding eating episodes. The significant positive effect of the quadratic function of time by LOC interaction term (estimate = 54.61,  $p = 0.03$ ) reflects the steeper decline in HR characterizing LOC episodes, as compared to non-LOC episodes, in the approximate 2.25 – 1 hour preceding the episode (Figure 14). Overall model  $R^2 = 0.07$  and  $\Delta R^2$  for LOC and LOC interactions = 0.06.

For SDNN, there was a significant main effect of episode type, suggesting that at the time of the eating episode, LOC status was predicted by significantly lower SDNN (estimate = -79.42,  $p = 0.04$ ). Linear, quadratic, and cubic effects of time were not

significant ( $p$ 's  $> 0.49$ ), but there were significant interaction effects between episode type and time functions, suggesting differences in trajectories of SDNN prior to LOC versus non-LOC episodes. The significant negative estimate of the cubic time function by LOC interaction term (estimate = -22.24,  $p = 0.02$ ) reflects the greater rate of decline in SDNN within the approximate 3 to 2.25 hours prior to LOC episodes compared to episodes not characterized by LOC. The corresponding rise in SDNN characterizing the approximate 2.25 – 1 hour prior to LOC, but not non-LOC, eating episodes, is represented by the significant negative estimate for the quadratic function of time by LOC interaction term (estimate = -105.51,  $p = 0.03$ , Figure 14). Overall model  $R^2 = 0.04$  and  $\Delta R^2$  for LOC and LOC interactions = 0.04.

Trajectories of parasympathetically-mediated HRV prior to eating episodes were examined via three indices: RMSSD, pNN50, and normalized HF-HRV. RMSSD and pNN50 elucidated similar findings. For both indices, no main effects were seen for any time functions. Main effects for LOC, wherein LOC episodes were immediately preceded by significantly lower RMSSD (estimate = -127.03,  $p < 0.001$ ) and pNN50 (estimate = -33.02,  $p < 0.01$ ) were observed. Similar to HR and SDNN, interaction effects between episode type and time functions suggested differences in trajectories of RMSSD and pNN50 based on episode type. As evidence by significant negative estimates for the cubic function of time by LOC interactions and the quadratic function of time by LOC interactions, LOC episodes were preceded by steeper declines in RMSSD (estimate = -38.59,  $p < 0.001$ ) and pNN50 (estimate = -10.11,  $p < 0.001$ ) approximately 3 - 2.25 hours prior to eating, followed by steeper inclines in both indices thereafter (for RMSSD estimate = -185.33,  $p < 0.001$ ; for pNN50: estimate = -48.48,  $p < 0.001$ ). There

were also significant interactions at the linear level, suggesting that within the 1 - 0.5 hours prior to eating, there were steeper declines in RMSSD (estimate = -245.70,  $p = 0.001$ ) and pNN50 (estimate = -66.21,  $p < 0.01$ ) prior to LOC, compared to non-LOC, episodes (Figure 14). For RMSSD, overall model  $R^2 = 0.09$  and  $\Delta R^2$  for LOC and LOC interactions = 0.08. For pNN50, overall model  $R^2 = 0.08$  and  $\Delta R^2$  for LOC and LOC interactions = 0.07.

For normalized HF-HRV, a similar pattern of findings was observed, wherein the LOC curve depicted a steeper decline and corresponding rise in HF-HRV prior to LOC as compared to non-LOC episodes yet no significant differences were observed based upon episode type ( $p's_{(LOC \times time^2, LOC \times time^3)} \geq 0.26$ ). For normalized HF-HRV, however, linear (estimate = -0.38,  $p = 0.02$ ), quadratic (estimate = -0.26,  $p < 0.01$ ), and cubic (estimate = -0.05,  $p < 0.01$ ) effects of time were significant, suggesting that all eating episodes were characterized by a decline, followed by a rise, followed by a second decline, in HF-HRV in the 3 hours preceding the eating episode (Figure 14).  $R^2 = 0.03$  and  $\Delta R^2$  for LOC and LOC interactions = 0.02.

### **Summary**

Figures 15 - 17 provide summaries of results for negative affect from mediation paths a – c for all data, and data used in the mediation model, respectively. For negative affect, examination of path a revealed that greater interpersonal problems generally predicted greater negative affect, producing a significant effect for the mediation model (Figure 15b) and a marginally significant effect for all data (Figure 15a). Findings from path b suggest that negative affect, but not immediate increases in negative affect, predicted LOC eating. Finally, for path c, interpersonal problems were predictive of

LOC eating in both models. For boredom, paths a and c revealed that interpersonal problems predicted boredom as well as LOC eating. For path b, boredom did not appear to predict LOC eating when all cases were examined. Similar to negative affect, examination of cases including all interpersonal model components suggested that baseline level of boredom, but not increase in boredom, predicted LOC. Finally, for positive affect, no significant relationships were found between interpersonal problems and positive affect or between positive affect and LOC eating.

***Hypothesis 3d.***

Negative affect will mediate the relationship between interpersonal problems and LOC eating. In other words, the correlation between interpersonal problems (X) and LOC eating (Y) will be significantly reduced when controlling for negative affect (M).

When considering whether mediation should be tested, paths a, b, and c must be significant. Because the change in state affect is examined as the dependent variable for path a, it is necessary that the change in state affect be considered the independent variable for path b. Although state affect (negative affect, boredom) were found to be significant predictors of LOC eating, changes in state affect were not. Therefore, based on the following results of hypotheses 3a – 3c for negative affect: interpersonal problems predicted LOC eating (path c); interpersonal problems predicted an increase in negative affect (path a); increase in negative affect did not predict LOC eating (path b), full mediation was not conducted for negative affect. Similarly, for boredom, path b was not significant and therefore mediation was not carried out.



## CHAPTER 5: DISCUSSION

### AIM ONE

To investigate whether self-report and physiologic EMA strategies are feasible and acceptable methodologies for examination of LOC eating behavior among adolescent girls with LOC eating, we recruited adolescents with recurrent LOC to complete an EMA protocol. The first aim of the study was to examine study feasibility and acceptability by measuring study compliance, attendance and acceptability ratings.

The average compliance rate among adolescents in the current study was nearly 70%. This is comparable to rates observed among samples of adolescents and children who participated in prior EMA studies of binge and LOC eating (81; 94), as well as EMA studies of other health behaviors among adolescents (e.g.'s (84; 127)) but lower than rates seen in adult studies of binge eating and bulimia nervosa (188; 194). In adolescent and child studies similarly assessing eating behavior, compliance rates within the range of 71 – 74% have been reported (81; 94). Among studies examining other physical and psychological health issues in adolescents, including diabetes treatment adherence (143), smoking cessation (84), post-concussive symptoms (127), self-injury (149), and peer relationships, affective states, and substance use (172; 236), a wider range of compliance rates—between 59% and 94%—was noted. By contrast, in two adult studies examining the role of negative affect in predicting binge eating and bulimic behaviors, higher compliance rates of 93% (194) and 86% (188) were reported.

For both eating related and other types of EMA studies among adolescents, varying compliance rates across studies may relate to differences in several methodological issues that may impact compliance. Such factors include length of the



study monitoring period, strategies used to monitor behavior in ambulatory settings (e.g. school involvement, use of cellular telephones versus palm top computers), strategies undertaken during the study to promote compliance (e.g. contacting participants if they miss EMA recordings), and reinforcements (e.g. payment) provided to youth for compliance.

Among 158 community adolescents asked to make random and event-contingent (before- and after-eating) reports regarding eating behavior over seven days, average compliance was 71% (81), similar to that observed among adolescents in the current study. However, adolescents in the study by Grenard and colleagues reported 1.92 eating episodes per day, above the average daily number of after-meal episodes (1.2) reported by adolescents in the current study. In the study by Grenard and colleagues, it is possible that compliance with event-contingent recordings was enhanced by teaching adolescents to upload their data from home and providing feedback to non-compliant adolescents about missed recordings on the day immediately following the missed recordings. Specifically, adolescents in the Grenard study were contacted by the study team if they missed data transfer at the end of the day, reported less than three eating/drinking events on the previous day, missed more than two random recordings in the previous two days, or missed two consecutive random prompts (81). Providing feedback early in the protocol enabled the study team to intervene more quickly when adolescents were non-compliant. In contrast, adolescents in the current study received feedback during twice-weekly or weekly meetings at the study site. As a result, cases of poor compliance were often undetected for several days. Futures EMA studies in adolescents with LOC eating should likely involve an earlier check-in with feedback to enhance adherence.

Another methodological feature of EMA research that may impact study compliance is whether participants are asked to complete event-contingent recordings before and after a target behavior occurs or after the target behavior occurs only. Adolescents participating in the study by Grenard and colleagues and children who participated in the study by Hilbert and colleagues were asked to complete a single rating after eating. By contrast, adolescents in the current study completed two ratings per eating episode, one before, and one afterwards. It is possible that requiring multiple ratings per meal increased participant burden and thereby caused adolescents to miss more random and event-contingent recordings throughout the monitoring protocol. Methodological decisions in EMA studies must balance the importance of information acquired during event-contingent recordings collected before the target behavior (in this case, during before-meal ratings) with the ability to gather this information in other ways (e.g. during random recordings occurring throughout the day) and the impact of requiring extra event-contingent recordings on overall compliance.

Populations of adolescents studied using EMA methodology range from clinical populations—such as those with ongoing medical or psychological conditions such as type 1 diabetes, post-concussive symptoms, self-injurious thoughts and behavior, and ADHD—to community samples of teens drawn from public school settings. Many studies have required adolescents to carry monitoring devices throughout the school day, and in some cases, such studies have solicited schools' involvement in order to facilitate students' participation. For example, one study enrolled three teens with post-concussive symptoms who were asked to complete EMA recordings five times per day for five days (127). In this study, each teen was granted permission to collect data during the school

day and alarms provided an alert every five minutes until the recording was completed by the teen. The compliance rate in this study, 93.3%, suggests that engendering school cooperation by granting students permission to participate in the study and allowing students to use the devices during school hours facilitates greater compliance. In contrast, in a study using twice-daily inbound cellular telephone calls to examine diabetes management adherence, teens answered or returned the call on only 59% of occasions (142). In the study by Mulvaney and colleagues, about one-third of teens indicated that they were allowed to use their cell phone during school, and 25% of teens indicated that cell phone access at school negatively affected how they manage their diabetes.

In the current study, teens were not asked to complete ratings during school, and they were advised not to bring the palm top computer to school in order to prevent losing the device. However, it is possible that leaving the palm pilot at home contributed to lower compliance, especially on days when teens participated in activities immediately following school. Indeed, teens in the current study indicated that one of the most common reasons for non-compliance was not having the device with them, and some teens indicated that they could not respond to the device while at school or after school activities.

Administration of recordings via cellular phones may constitute a preferred modality for data collection among many adolescents. Cellular phones may therefore enhance compliance for a number of reasons. First, cellular phones are popular among youth, and interacting with cellular phones is an activity that is familiar to many adolescents (222). Furthermore, teens may feel less self-conscious about using cellular telephones as compared to palm top computers in front of others. In addition to the

socially-relevant benefits of cellular phones, it is possible that fewer technological problems would be encountered because such devices are newer and more reliable. Data transfer may immediately follow data entry and therefore the potential for data loss between completion of the recording and upload is minimized.

Also relevant to conducting EMA research in pediatric samples is that youth may have less control over their schedules compared to adults. Whereas adults may be free to bring their EMA device wherever they travel throughout the day, youth may be restricted by school rules or limitations inherent to settings such as church, sports practices or other activities. Thus, greater structure and external environmental requirements may contribute to the observed difference in compliance rates between EMA studies of eating behaviors in adults versus youth. Additionally, it is possible that cognitive or developmental factors, such as executive function or maturity, may enable adults to achieve higher compliance with the daily requirements of EMA protocols. Indeed, the normative developmental course of complex cognitive processes associated with pre-frontal cortical maturity such as executive function continues through late adolescents into early adulthood (11). Executive functioning may relate to adults' compliance via its impact on planning ability, (e.g. enabling one to think ahead and bring the device to one location because they know they will not return home prior to going to a second location), organization, and prioritizing multiple tasks. Supporting the importance of executive functioning in enabling adolescents to successfully comply with the EMA protocol, adolescents in the current study commonly cited "not having the device with me" as a reason for missing recordings.

Another component of study feasibility is participants' willingness to attend study visits. All participants in the current study who agreed to complete the data collection phase of the study attended at least four study visits. Furthermore, over 90% of study visits were attended, with only three rescheduled visits that resulted in adolescents spending greater than seven days between consecutive study visits. Given the high rate of study attendance, we conclude that, consistent with the vast majority of child (94) and adult (188; 233) EMA research conducted to date, study attendance did not impede completion of the EMA protocol.

No child or adolescent study that we know of has examined the acceptability of using EMA to study LOC eating behavior among adolescents. Few adult studies in the eating disorders literature have reported on the acceptability of EMA protocols. The level of acceptability reported by adolescents in the current study was generally high. Although the study was designed with the intent of minimizing burden, adolescents characterized their self-monitoring experiences as mild to moderately disruptive and mild to moderately time consuming. Nonetheless, adolescents reportedly experienced the self-monitoring as helpful and positive, suggesting that the procedure was generally acceptable to adolescents. In studies examining the feasibility of EMA to examine other health issues among children and adolescents, results similarly suggested generally high rates of study acceptability. In a study of post-concussive symptoms among three adolescents, youth participated in debriefing interviews and reported that study procedures were not problematic at a data collection rate of five times per day for five days (127). In a child study using EMA to examine physical activity and sedentary behaviors among youth, eight children participated in interviews during a pilot phase of



the study, and six out of eight children reported that the phone was easy to use and unobtrusive, and all children reported that they would participate in a future EMA study of a similar nature (49).

To our knowledge, no study has examined the acceptability of using EMA to study LOC eating behavior among adolescents. Few adult studies in the eating disorders literature have reported on the acceptability of EMA protocols. The level of acceptability reported by adolescents in the current study was generally high. Although the study was designed with the intent of minimizing burden, adolescents characterized their self-monitoring experiences as mild to moderately disruptive and mild to moderately time consuming. Nonetheless, adolescents reportedly experienced the self-monitoring as helpful and positive, suggesting that the procedure was generally acceptable. In studies examining the feasibility of EMA to examine other health issues among children and adolescents, results similarly suggested generally high rates of study acceptability. In a study of post-concussive symptoms among three adolescents, youth participated in debriefing interviews and reported that study procedures were not problematic at a data collection rate of five times per day for five days (127). In a child study using EMA to examine physical activity and sedentary behaviors among youth, eight children participated in interviews during a pilot phase of the study, and six out of eight children reported that the phone was easy to use and unobtrusive, and all children reported that they would participate in a future EMA study of a similar nature (49).

A final aim in evaluating study feasibility was to assess the impact of demographic and anthropometric variables, as well as variables of interest, on dimensions of study feasibility including compliance and acceptability. Consistent with study



hypotheses, demographic and anthropometric factors including age, race, height, and percent adiposity were all unrelated to adolescents' compliance and acceptability ratings. In contrast to study hypotheses, several relationships between dimensions of compliance and acceptability and study-related variables were found. Interpersonal problems and LOC eating were found to be associated with lower study compliance. Negative affect was also found to be marginally associated with poorer compliance, but this relationship was tempered when outlier values were excluded, suggesting that the finding was driven in part by outlier values. Negative affect was marginally related to lower study acceptability, before and after accounting for outlier values. Finally, LOC eating initially appeared to predict poorer acceptability, but this effect was no longer observed when outliers were excluded.

There are several potential explanations for the observed associations between study-related variables, including interpersonal problems, negative affect, LOC eating, and the feasibility dimensions of compliance and acceptability. First, it is possible that negative affect may reduce compliance and acceptability via mechanisms of motivation and anhedonia, associated with depression. It is possible that negative affect serves as an underlying construct producing fatigue, reduced energy, and anhedonia, causing adolescents to be less compliant with study procedures and rate their experiences of the study less positively. In studies using EMA methods among depressed youth, compliance rates are generally good (16; 184), however, it is possible that among a sample non-homogenous for depressive symptoms, those with greater symptoms may be less compliant.

It is also possible that individuals with greater negative affect may lack adequate social support. Negative associations were noted between interpersonal problems and dimensions of feasibility, such that adolescents who reported more interpersonal problems had lower compliance and acceptability ratings. Thus, social support may serve as another potential mediating variable between negative affect and feasibility dimensions. Social support may also directly impact feasibility in a number of ways, such as via parental encouragement or peer support. To our knowledge, no study has directly tested relationships between social support or interpersonal problems and dimensions of EMA feasibility such as compliance and acceptability. However, data from literature examining the relationship between social support and adherence to medical and behavioral health regimens suggests that there is a robust relationship between adherence to medical treatment and social support among youth and adults (46). In a meta-analysis by DiMatteo, 2004, adherence was associated with several facets of social support including practical support (e.g. reminders, organization, and support for a specific behavior), emotional support (e.g. nurturance), overall social support, and family cohesion (e.g. warmth, acceptance, and closeness) (46). Among youth, having married parents was also associated with greater treatment adherence. Several adolescent studies have examined associations between dimensions of parental support and adherence to type 1 diabetes regimens, with findings generally indicating a positive relationship between parental monitoring and involvement and treatment adherence (10; 55). Although the current study involved compliance with a study-related EMA procedure, rather than adherence to a medical regimen, it is possible that adolescents whose parents encouraged and supported their study involvement may not only rate their interpersonal

problems as lower, but achieve higher compliance due to parental support. Similarly, adolescents who perceive positive peer relationships may be more open about their study involvement and be more likely to complete palm pilot recordings in front of peers. These teens may rate the study as interfering with day-to-day activities to a lesser degree, producing higher scores on the dimension of acceptability.

Analogous to the relationships detected between interpersonal problems and negative affect and dimensions of feasibility, LOC eating was inversely related to study compliance. The observation that adolescents with greater LOC eating are less compliant may be influenced by social desirability or related constructs. It is possible that adolescents with more frequent LOC eating may be less compliant with EMA monitoring due to greater feelings of self-consciousness related to LOC episodes as compared to adolescents who experience less frequent episodes. In other words, the construct of social desirability—hypothesized to cause adolescents to make fewer EMA recordings in which they are queried about their eating behavior—may operate more strongly among teens experiencing greater episode frequency. Although no study has directly examined the relationship between social desirability and the discrepancy between actual and reported LOC episodes, one adult study found that women with higher scores on a measure of social desirability were more likely to underreport their dietary intake (174).

Despite associations between dimensions of the interpersonal model and compliance and acceptability, aims two and three were still able to be examined. Overall compliance was still in the acceptable range and only two adolescents' compliance rates were lower than 50%. Overall acceptability ratings were generally high.

## AIM TWO

The second aim of the current study was to examine the concordance of two methods for assessing the affective component of the interpersonal model: (1) self-reported negative affect (assessed via questions administered via palm top computers) and (2) a physiologic proxy for negative affect, HR and HRV, a constellation of indices thought to elucidate autonomic regulation and regulatory capacity. We hypothesized that negative affect would be correlated positively with HR and negatively with HRV, and the relationship between negative affect and overall HRV was posited as an exploratory aim.

Findings at the trait level revealed a marginal positive association between trait anxiety and HR and a marginally negative association between total psychiatric symptoms and mood and internalizing symptoms and HF HRV. At the state level, daily negative affect was inversely associated with HF HRV. No other relationships were observed between indices of state affect and HR and HRV. At the momentary level, relationships were observed between negative affect and two physiologic indices. Adolescents' reports of greater momentary negative affect were associated with greater HR and marginally lower pNN50.

Several studies have examined the relationship between trait and state affect and HR and HRV. The marginal association between depressive symptoms as measured by clinical interview and lower parasympathetically-mediated HRV is consistent with adult depression literature, in which this association is generally well-accepted (108; 231). Similarly, although few adolescent studies have examined HR and HRV indices in relation to depression, a small study among adolescent girls comparing those with and without major depressive disorder found that girls with major depression had lower

indices of parasympathetically-mediated HRV as measured in a laboratory paradigm (223).

In the current study, greater mood and internalizing symptoms reported during an interview with a clinician, but not on a self-report measure, were associated with reduced HF HRV. The discrepancy in the relationship between HF HRV and depressive symptoms when findings were assessed based on interview versus self-report measures was unexpected. It is possible that the two methodologies used in the current study to assess depressive symptoms captured slightly different constructs or that varying statistical properties of each measure resulted in different findings. Whereas the BDI-II produced a normal distribution of depressive scores around a mean of mild depression, the KSADS captured greater depressive symptoms in the sub-clinical and clinical range, and most adolescents in the study did not experience depressive symptoms sufficient to produce a score greater than 0 or 1 on the KSADS. Furthermore, whereas the KSADS and other clinical interviews are intended to diagnose clinical depression and sub-clinical depressive symptoms, the BDI and similar questionnaire measures are intended for use as screening instruments (20). Literature from the eating disorders field posits that interview methodologies constitute a preferred assessment method, as interviews garner more accurate information, provide the opportunity to properly define symptoms, and reduce the incidence of false positives and negatives (209).

It is possible that the relationship between depression and HRV is non-linear, such that HF HRV is unaffected below a specific threshold, above which depressive symptoms are inversely associated with HF HRV. Such a relationship would potentially enable a pattern to be detected between KSADS scores and HF HRV, but not between



BDI-II scores and HF HRV. In support of this hypothesis, an adult study examining HR and HRV indices among healthy controls and moderately and severely depressed patients found that lower HF HRV was present only among the severely depressed patients. Furthermore, two indices of HRV, including RMSSD, were significantly lower among the severely, as compared to the moderately, depressed subjects (2). It is also possible that this finding is the result of Type I error, producing a false positive finding, and that the relationship is not truly present among adolescents with LOC. Additional, adequately-powered analyses are needed in order to fully explore this relationship.

Associations between negative affect and HR and HRV indices were also examined at the daily and momentary levels. Results revealed that both daily negative affect and momentary negative affect were either significant or marginally associated with lower HF HRV. No ambulatory study in adolescents, and few among adults, has examined the role of daily state affect on physiologic stress indices. Among a sample of cardiac patients, Bhattacharyya and colleagues found that aggregate daily positive affect, assessed retrospectively via the daily reconstruction method, was associated with greater parasympathetic autonomic modulation independent of the contribution of depression (22). Momentary influences of state affect, as well as other constructs, on autonomic indices are more often examined in laboratory, as compared to EMA, paradigms. One adult study examining autonomic indices in response to happiness and anger inductions found that participants who experienced happiness in response to the happiness induction demonstrated an increase in HF HRV. Conversely, those who experienced an increase in frustration during the happiness induction demonstrated a decrease in HF HRV. During the anger induction, greater levels of frustration were marginally associated with



increases in LF HRV, and feeling “in control” and “interested” were protective against increases in the LF:HF ratio (115). In an adolescent study that examined HF HRV in response to an anger induction among adolescents categorized as “reappraisers” or “ruminators,” based on their responses to the induction paradigm, reappraisers were found to have greater HF HRV at baseline and recovery. Consistent with the current study’s finding that negative emotions were marginally associated with HF HRV in the natural environment, results of the studies noted above suggest that the negative emotions (e.g. frustration) are potentially associated with relative decreases in HF power. Taken together, findings collectively suggest that negative emotional states may be accompanied by lower capacity for autonomic control of the heart. In the current study, no association was observed between positive affect and HF HRV, which is in contrast to previous literature (22; 115). The absence of an association in the current study may result from differences in study design, sample characteristics, or study methodology (e.g. methods used for assessing positive affect)..

Given the pilot nature of the study and the small sample size, marginally significant findings were reported even though they should be interpreted cautiously and as hypothesis-generating. Nevertheless, the observed associations between negative affect and HF HRV were relatively consistent at all temporal levels, and aligned with a priori study hypotheses positing an inverse association between depression and negative affect and lower parasympathetically-mediated HF HRV. In future studies, testing of relationships within an adequately powered design is necessary to draw definitive conclusions about the relationships between psychiatric and mood symptoms and daily

and momentary affective states as they relate to autonomic indices of HR and parasympathetically-mediated indices of HRV.

### **AIM THREE**

The final aim of the study was to conduct a preliminary investigation of the interpersonal model of LOC eating among adolescent girls. Accordingly, unique relationships between interpersonal problems and state affect, state affect and LOC eating, and interpersonal problems and LOC eating were examined in two types of mixed models, one including all available data, and a second including only cases that included an interpersonal problem rating, a state affect rating, and an eating episode (comprising all data points needed to evaluate the interpersonal model). A mediation model, wherein negative affect was proposed to mediate the relationship between interpersonal problems and LOC eating, was proposed.

The first path proposed by the interpersonal model was the link between interpersonal problems and LOC eating. For this link, results from both mixed models indicated that interpersonal problems were a strong predictor of LOC eating. The second path examined was the link between interpersonal problems and state affect. For this path, results from first set of mixed model analyses suggested that interpersonal problems predicted increases in boredom, marginal increases in negative affect, and were unrelated to positive affect. Examining cases included in the interpersonal model only, interpersonal problems predicted negative affect and boredom, but not positive affect. The third path in the interpersonal model was the link between state affect and LOC eating. In the first set of models examining level of state affect only, LOC eating was

predicted by higher level of negative affect, but not by boredom or positive affect. In the second set of models including level of state affect and the change in state affect, higher negative affect predicted LOC eating, but the change in negative affect did not.

Similarly, for boredom, higher level of boredom, but not change in boredom, predicted LOC eating. For positive affect, neither level of positive affect nor the change in positive affect significantly predicted LOC eating. Effect sizes detected for relationships between interpersonal model variables ranged from negligible (for the relationship between negative affect and LOC) to small (for the relationship between interpersonal problems and LOC).

The relationships between interpersonal model variables were also tested using a physiologic index of negative affect. Results suggested a moderately strong link between interpersonal problems and physiologic indices. Compared to times of “low” interpersonal problem ratings, adolescents had higher HR and lower parasympathetically-mediated HRV at times of “high” interpersonal problem ratings. Although there were few differences in the trajectories of HR and HRV following “high” versus “low” interpersonal problem ratings, there was a modest difference in the slope of the HF-HRV curve following high versus low interpersonal problems. Differences in trajectories of physiologic indices prior to LOC versus non LOC episodes following LOC and non-LOC episodes were also examined, and results suggested that LOC and non-LOC episodes are characterized by significant differences in the trajectories of HR and HRV prior to the eating episode. In general, LOC episodes were often preceded by relatively steeper increases in HR and decreases in parasympathetically-mediated indices of HRV, followed by corresponding steeper decreases in HR, and increases in HRV indices, within

the several hours preceding the eating episodes. Findings remain extremely preliminary; however, this pattern is consistent with an autonomic stress response.

Overall, study data lend support to the hypothesis that interpersonal problems may contribute to small, significant variability in LOC eating. Our data are among the first to directly test links between interpersonal factors and LOC eating in an ambulatory setting. Another child study that examined the link between interpersonal factors and LOC eating examined differences in dimensions in family functioning by LOC status using coded videotaped recordings of family mealtime interactions among 8 – 13 year old children with and without LOC eating in families' home settings. Study findings indicated that families of children with LOC demonstrated lower overall family functioning, particularly in the domains of interpersonal involvement and communication, relative to families of children without LOC (38). The majority of prior research examining the hypothesis that interpersonal factors are associated with LOC eating has been conducted in the laboratory, via designs examining food intake (rather than LOC specifically) in response to interpersonal stressors. For example, among 8 – 13 year old children, critical comments by parents during a laboratory test meal were associated with greater energy intake during a subsequent snack eating period (96). Adult research similarly suggests that stressors of an interpersonal nature may predict increased subsequent food consumption in a laboratory setting (60; 152). Our findings are consistent with these prior laboratory studies and fill a needed gap in our knowledge among adolescents. These preliminary data support the proposal that interpersonal stressors play an important role in the etiology and maintenance of LOC eating.

The activation of physiologic stress pathways by interpersonal stressors, such as negative family interactions or peer rejection, may constitute one mechanism by which interpersonal problems are associated with subsequent changes in LOC or eating behavior. In the current study, significant aberrations in autonomic functioning—including higher HR and lower parasympathetically-mediated HRV—were observed at times when adolescents reported high, as compared to low levels of interpersonal problems. In a laboratory study, Epel and colleagues demonstrated that the relationship between exposure to a laboratory stressor and food intake was moderated by physiologic stress reactivity. In this paradigm, women participated in two laboratory sessions, one consisting of a social stressor, and the other consisting of a control condition. Cortisol reactivity, a measurement of stress responding, was measured on both days. Findings indicated that while women with high cortisol reactivity ate significantly more relative to women with low cortisol reactivity following exposure to the stressor, women consumed similar amounts following the control condition (60). Findings from the current study suggest that such a mechanism, wherein stressors of an interpersonal nature may induce a physiologic stress response, promoting LOC eating, may operate within the natural environment.

The finding that negative affect is predictive of greater subsequent LOC is generally consistent with adult EMA literature suggesting that negative affect is greater on binge, compared to non-binge days (188; 233) and at binge, compared to non-binge, times (95). Findings are also consistent with laboratory data among adolescents suggesting that pre-meal negative affect promotes greater consumption of highly-palatable foods (161). In contrast to EMA data among adult patients with bulimia



nervosa (21; 188), data from the current study did not support the hypothesis that LOC episodes are preceded by an immediate increase in negative affect. Furthermore, in contrast to the strong links observed between interpersonal problems and LOC eating, the relationship between negative affect and LOC eating was no longer significant when interpersonal problems were accounted for in the model. Nor did negative affect appear to mediate the relationship between interpersonal problems and LOC eating based on the current test of the interpersonal model.

The absence of an association between negative affect and LOC eating when accounting for interpersonal problems is partially in concert with existing child data regarding the relationship between negative affect and LOC eating. Minimal evidence among preadolescents supports a relationship between these two constructs. In the only other examination of LOC among youth (8 – 12 years) using an EMA paradigm, Hilbert and colleagues (94) concluded that affective factors did not play a causal role in LOC eating among youth. Furthermore, in the laboratory paradigm noted previously (96), children's pre-meal mood ratings did not significantly predict eating behavior, in spite of the fact that youth ate more following critical comments by parents.

Taken together, there are several potential explanations for the pattern of associations between interpersonal problems, negative affect, and LOC eating. First, interpersonal problems may simply be a more salient predictor of eating behavior among adolescents, as compared to self-reported emotional states. It is possible that ability to identify and report one's emotional state may follow a developmental trajectory, which may explain why negative affective theories of LOC are generally not supported among children (94; 95) yet receive robust support when tested among adults (e.g. (85)).



A related hypothesis is that the construct of alexithymia, comprising difficulty identifying and reporting one's emotional state and having a cognitive and externally oriented thinking style, may moderate the relationship between state affect and LOC eating among youth with LOC. Preliminary data from the Toronto Alexithymia Scale-20, a 20-item self-report measure of alexithymia administered to adolescents in the current study prior to initiation of the EMA monitoring period, revealed that adolescents with higher scores on the TAS Externally Oriented Thinking subscale reported marginally lower average negative affect over the assessment period. However, average negative affect was not associated with scores on subscales related to identifying or describing emotions. Preliminary examination of this measure also revealed that greater impairment on the Externally Oriented Thinking subscale was associated with less variability in negative affect over the monitoring period. Such findings lend preliminary support to the notion that adolescents' capacity to reflect on their emotional experiences may influence the relationship between negative affect and LOC eating. Additional analyses examining the moderating role of alexithymia may aid in clarifying relationships between affective states and LOC.

Studies demonstrating the most robust relationships between negative emotions and binge and bulimic behaviors (e.g.'s (95; 188; 194) have been conducted among clinical samples of adults with full-syndrome eating disorders, whereas the majority of adolescents in the current study, as well as children included in the study by Hilbert and colleagues, represent youth with sub-clinical presentations. Progression of pathological dysregulated eating behavior from infrequent LOC episodes to full syndrome BED may negatively reinforce the utility of LOC and BE for affect regulation.

It is also possible that our chosen measure (the PANAS) limited our ability to detect emotional states contributing to aberrant eating behavior among youth. The PANAS, assessing such emotional states as happy, sad, delighted, and upset, includes a range of emotions of fairly high negative and positive valence. Measures geared toward the detection of anxiety or other, more subtle perturbations in emotional state (irritability, agitation), may be more likely to detect the types of emotions contributing to LOC eating among youth.

Although self-reported indices of negative affect were only marginally predictive of LOC episodes, examination of autonomic trajectories prior to LOC and non-LOC episodes revealed significant physiologic differences based upon episode type. Effect sizes for physiologic findings were greater than for self-report findings, suggesting that LOC status explained a small to medium proportion of variability in physiologic indices preceding the episode. If analyses within adequately powered designs are confirmatory, it is possible that LOC and non LOC episodes may be differentiated by the autonomic patterns observed prior to the episode. LOC episodes were characterized by autonomic reactivity followed by partial recovery, a pattern suggestive of a biologic stress response, whereas non-LOC episodes were characterized by relatively stable HR and HRV trajectories. Cardiovascular responses like those observed prior to LOC episodes are commonly elicited by child and adult laboratory studies in which stressors of a cognitive, emotional, or interpersonal nature are employed (e.g. (133; 175; 230)).

Autonomic reactivity and recovery were observed to occur approximately two hours, rather than immediately, prior to the relevant eating episode. It is possible that youth may experience a stressor and subsequent stress response but lack the opportunity

to eat, resulting in the experience of LOC eating much later. Alternatively, LOC youth may experience a stressor and lack appropriate coping skills, resulting in lingering stress, and ultimately promoting LOC eating several hours later. These data suggest that LOC eating may be a prolonged consequence of physiologic stress, and are consistent with our self-report data suggesting that LOC episodes are not precipitated by immediate increases in negative emotional states.

There are several potential mechanisms by which changes in HR and HRV may promote LOC eating. First, it is possible that individual with LOC may experience greater physiologic reactivity in response to stressors, compared to those without LOC. In support of this explanation, Friederich and colleagues demonstrated that BED subjects experienced augmented reductions in HF HRV (greater vagal withdrawal) in response to a mental task during a laboratory study examining HF HRF reactivity and recovery among adult women with BED versus control subjects (69). In this study, the authors concluded that greater parasympathetic withdrawal in response to a stressor suggested greater stress vulnerability among the BED group.

In contrast, an opposing hypothesis suggests that those with LOC eating (or other psychological problems) may exhibit a blunted response when exposed to stressors that would be expected to elicit a physiologic stress reactivity and recovery. In support of this notion, a study examining autonomic reactivity and recovery among individuals with BN, BED and control subjects found that only the control subjects experienced the expected decrease in HF HRV (vagal withdrawal) in response to the stressor, and a corresponding increase in HF HRV thereafter (136). In this study, the authors considered vagal withdrawal to be the adaptive physiologic response, and concluded that BED patients

demonstrate limitations in their capacity for adaptive autonomic responding. The latter approach is consistent with theories of restricted autonomic responding (221), in which tonic hyperarousal of the PNS is thought to blunt the window of capacity of the PNS to respond to environmental stressors. Limited capacity for adaptive autonomic response to stressors may impair adaptive recovery following stressors that elicit autonomic reactivity in the natural environment. Supporting this hypothesis, mixed models of the effects of time and eating episode type on HR and HRV in the current study suggest that autonomic indices may not return to baseline following reactivity, suggesting that autonomic recovery is only partial. It is possible that impaired recovery may prime individuals for LOC eating, in an attempt to regulate the body's internal milieu. Additionally, children and adolescents may have limited adaptive strategies for coping with interpersonal stressors that leads to only partial physiologic recovery. Indeed, one study among 8 – 13 year old children found that youth with LOC reported the more frequent use of maladaptive emotion regulation strategies, such as perseveration, relative to their non-LOC peers (39).

Another potential mechanism by which physiologic changes promote LOC may be via glucoregulatory processes. Autonomic activation (increase in HR, decreases in parasympathetically-mediated HRV) induces a number of physiologic processes that may prime the body for eating, via eventual production of insulin. Specifically, the reactivity phase of the stress response is characterized by a release of glucagon and epinephrine, which mobilize blood sugar via gluconeogenic and glycolytic processes in the liver, muscles and other areas of the body. Following the reactivity phase, recovery is in turn characterized by an opposing response including the release of insulin. Elevated insulin

concentration causes a reduction in blood sugar, which in turn promotes hunger, possibly priming the body for eating. Analyses addressing the contributions of pre-meal hunger to subsequent LOC and non-LOC episodes may aid in examining this hypothesis.

Differentiating whether and how glucoregulatory processes specifically promote LOC, as compared to non-LOC episodes, may further elucidate this potential mediating mechanism.

#### **CLINICAL IMPLICATIONS**

Although the current study was designed with the primary aim of establishing feasibility, acceptability, and refining hypotheses for a larger assessment study, clinical implications can also be informed by study findings. The current study's finding that interpersonal problems are a robust predictor of LOC eating among adolescent girls suggests the importance of targeting interpersonal factors among adolescents with LOC as well as elucidating mediators between interpersonal factors and LOC eating. Among adults with BED, IPT has been found to effectively reduce binge eating in the short- and long-term (92; 238) and a pilot study among adolescents with LOC eating demonstrated that girls randomized to the IPT group experienced greater reductions in LOC eating (213).

Contrary to study hypotheses, current study findings indicated that negative affect did not mediate the relationship between interpersonal problems and LOC. However, physiologic data indicating that heart rate is higher, and parasympathetic control, lower, at times of interpersonal distress, suggest that emotion regulation may play a role in the relationship between interpersonal problems and LOC. While additional research is



needed to clarify these relationships, interventions targeting emotion regulation may facilitate changes in physiologic responses to interpersonal stress, and in turn, promote reductions in LOC eating. Interventions targeting emotion regulation include biofeedback and psychosocial strategies such as those involved in dialectical behavior therapy.

The observed associations between model components (interpersonal problems and LOC eating) and dimensions of study feasibility also have important implications for EMA intervention research. Specifically, findings of the current study suggest that adolescents with the greatest impairment, as reflected by high levels of interpersonal problems and LOC eating, might exhibit the poorest compliance with a study intervention. Thus, intervention programs that accommodate non-compliance (e.g. do not require complete compliance in order to achieve an effect) may be ideally suited for adolescents with the most frequent and severe interpersonal problems and LOC eating.

Finally, many study participants reported during the study debriefing interview that their awareness of their eating patterns increased during the study period, which many participants attributed to daily monitoring of variables using electronic devices. This data provides anecdotal support for the utility of daily monitoring during an intervention protocol. Additional research would be needed to determine the relative influence of additional factors including external accountability and monetary compensation, in addition to simply tracking one's mood and eating patterns.

While the study was designed to recruit a diverse sample, African American adolescents comprised a disproportionately high percentage of the study sample (63.3%) in comparison to the proportion of African Americans (27.2%) comprising the D.C.



metropolitan population. This is consistent with the recognized health disparity in obesity rates, with higher rates of obesity seen among African American and other minority populations as compared to Caucasians (150). Therefore, the design of interventions that are acceptable among African American youth is particularly important, and the current study provides preliminary feasibility data regarding naturalistic research.

#### **STRENGTHS AND LIMITATIONS**

The current study was the first to examine interpersonal and affective predictors of LOC eating in the natural environment among adolescent girls with LOC eating. The overarching objective of this study was to evaluate the feasibility and acceptability of conducting a larger naturalistic study of interpersonal and affective predictors of LOC episodes. Strengths of the study include the multimodal assessment of adolescent's emotional experiences via self-report and physiologic dimensions; the use of gold-standard assessment methodology, including validated and reliable measures for assessing eating pathology (the Eating Disorder Examination), psychopathology (the Kiddie Schedule for Affective Disorders and Schizophrenia), and body composition (air displacement plethysmography); and the inclusion of a racially- and ethnically-diverse sample. Another strength of the study was the use of EMA, improving ecological validity and generalizability of findings prior retrospective (54) and laboratory (161; 208) studies to adolescents' naturalistic settings.

The proposed study had several limitations. Although the study was a pilot by design, such studies typically include small, homogeneous samples. Indeed, we enrolled only overweight, adolescent girls. While this strategy decreased variability,

generalizability to individuals not included in the study sample is limited. We restricted the sample to adolescent girls because girls are thought to be more sensitive to interpersonal distress compared to boys (200), and because girls exhibit higher rates of LOC eating during adolescence (65; 148). Because boys were not included in the study, conclusions regarding the feasibility of study procedures among adolescent boys cannot be determined, nor can the relevance of the interpersonal model for boys be inferred. Additionally, the present study only included overweight adolescents, despite data suggesting that some adolescents of a healthy weight status experience LOC eating (215). Non-overweight individuals were excluded because LOC is more common among overweight youth (147; 148) and because the interpersonal model is thought to be of greater relevance for overweight adolescents, as overweight teens are at greater risk for interpersonal and social problems such as peer rejection and victimization (89; 153; 198). As a result, inferences regarding application of the interpersonal model to non-overweight adolescents cannot be made. Finally, individuals with major medical and/or major psychiatric illnesses were not included in the present study because it was considered unethical to include girls with DSM-IV-TR threshold diagnoses in a non-treatment study, rather than immediately referring these individuals for appropriate treatment. Study results, therefore, are not generalizable to those with major medical and psychiatric illnesses. Future research may aim to extend findings to these groups.

Furthermore, the size of our sample, while ideal for a pilot study aimed to examine feasibility, as proposed, was not adequately powered to examine aims two and three. Therefore, results of marginally significant values are discussed throughout. In spite of multiple analyses, no statistical correction was performed. This decision was

made in light of the small sample size and limited power, and because the primary objective of hypotheses two and three was to generate preliminary data for an adequately-powered study to test the interpersonal model.

The EMA protocol itself was designed to maximize sampling of the target behavior and relevant predictors (interpersonal events, state affect), while minimizing participant burden. Yet, a number of limitations, including inability to administer EMA procedures during school and assess features of eating episodes such as amount and types of foods consumed, constitute study limitations. Due to feasibility reasons, a significant study limitation was that adolescents were not queried about their experiences during the school day. This shortcoming is mitigated by data suggesting that LOC episodes generally occur after school hours (187; 207), yet it is possible that important eating episodes, interpersonal events, and mood shifts occur during the school day for some adolescents.

Additionally, the present study did not address factors related to adolescents' eating episodes including the amount and types of foods consumed by adolescents. Such information would aid in understanding the relevance of LOC episodes for related health problems such as excess weight gain among adolescent girls. However, research suggests that LOC, rather than the amount and type of food consumed, constitutes the key feature of pathological eating among youth (138; 215). Moreover, the meal composition of youth with LOC eating has been phenotyped in the laboratory (208), demonstrating that LOC meals typically consist of energy dense, palatable foods.

Finally, there are a number of limitations specifically related to collection of HRV data. HRV data collection is highly burdensome for participants because youth must

wear an ambulatory monitoring device for 48 hours, which often interferes with regular activities. Although adolescents were informed that they should engage in their usual activities (with the exception of vigorous activity and swimming), it is possible that days on which the monitor was worn were atypical (e.g., adolescents might have reduced or abstained from socializing or engaging in even mild physical activity during the time that they were wearing the monitoring equipment). Adolescents were also required to abstain from taking a shower for 48 hours while engaging in Holter monitoring. Furthermore, due to the burdensome nature of wearing a Holter monitor, adolescents were only asked to engage in Holter monitoring on two days out of the 14-day protocol, resulting in a limited number of days on which HR and HRV data was gathered. Due to the pilot nature of the study, technical problems interfered with successful collection of HR and HRV data, further decreasing the proportion of HR and HRV data collected.

As a result of these limitations, significant study findings regarding associations between physiologic indices and self-report variables should be considered in light of potential concerns regarding generalizability and the small sample, and are in need of replication with larger samples before definitive conclusions may be drawn. However, the emergence of significant effects in spite of the limited sample size suggests that such relationships warrant additional investigation. Assessment of HR and HRV using a device such as the Actiheart®, containing only two electrodes, that fits more comfortably underneath participants' clothing and/or is more easily able to be taken on and off (in order to bathe/shower) may enhance generalizability of physiologic data and may engender collection of HR and HRV data on a greater number of days per person, improving the statistical power of the study.

An additional limitation related to ambulatory monitoring of autonomic indices is related to the high number of visceral and external variables known to influence HR and HRV, including food intake, posture, movement, physical activity, speech patterns, and respiratory parameters. First, food ingestion may impact autonomic balance as previously described (70; 86). In one study, participants experienced a biphasic shift in low- to high-frequency ratio in response to a meal, with a relative increase in LF power during eating that quickly abated after eating. However, these shifts were not significantly different from pre-prandial low- to high-frequency ratio (86). To control for the impact of food intake, when recorded by participants, the impact of rating type (e.g. random, before-meal, after-meal) was examined as a potential covariate in relevant analyses. In analyses of the trajectories of physiologic parameters preceding eating episodes, overlapping eating episodes were excluded in order to control for the influence of eating on HR and HRV. However, it is likely that participants missed some before- and after-meal recordings, meaning that these eating events were unaccounted.

Physical activity and body position also exert significant influence on HR and HRV. Perini and colleagues (1996) demonstrated that LF power is significantly increased, and HF power significantly reduced, in supine compared to sitting position (154). We therefore controlled for body position when possible using participant self-report of their body position (e.g. lying down versus sitting up). Although current analyses examined the potential influence of covariates such as posture and rating type, it is possible that additional factors may confound relationships between variables of interest and autonomic indices. For example, regarding the relationship between autonomic indices and LOC episodes, it is possible that participants demonstrating



autonomic reactivity and recovery prior to LOC episodes exercised prior to the eating episode, promoting gluconeogenic processes and subsequent LOC eating. Because the preliminary analyses of the current study did not account for posture/physical activity, these explanations cannot be ruled out.

Event-contingent recordings were not collected at the time of interpersonal events (but rather at the time of eating episodes), meaning that an adolescent's interpersonal problem rating might occur subsequent to her emotional and physiologic response to the event. Since adolescents reported interpersonal problems at the time that they were prompted, rather than at the time that the interpersonal problem actually occurred, it is not surprising that the trajectories of HR and HRV did not differ following high versus low interpersonal problems. Future research examining physiologic responses following interpersonal events would require that the EMA protocol include event-contingent prompts at the time of the perceived interpersonal problem (a conflict or feelings of loneliness).

Finally, linear mixed models, while ideal for circumventing many traditional problems of GLM, do not enable the partitioning of variance according to whether effects are derived from between- versus within- subject variability. Therefore, current analyses do not enable the determination of whether observed effects are significant at the between-subjects level (e.g. individuals with higher interpersonal problems exhibit greater LOC eating), the within-subjects level (e.g. individuals exhibit greater LOC when they experience greater interpersonal distress), or both. Therefore, additional analyses, such as those proposed by Hedeker and colleagues (2009), are needed in order to elucidate between- versus within-subjects variability within MLM (91). In brief,



Hedeker and colleagues describe that between- and within-subjects effects can be differentiated by including subjects' mean value for the independent variable (subject-level variable) in addition to a difference score (assessment level variable). A significant subject-level effect indicates a between-subjects effect, and a significant assessment-level effect indicates a within-subjects effect.

## CONCLUSIONS

With regard to the first study aim of evaluating the feasibility and acceptability of testing the interpersonal model of LOC eating among adolescent girls who report LOC, current study findings suggest that carrying out EMA among adolescents with LOC eating was feasible and acceptable. Adequate compliance, high attendance rates, and good acceptability ratings collectively suggest that adolescents with LOC and their families are able to engage in the procedures required for an EMA study design. The current study also elucidated potential strategies for enhancing compliance among adolescents, namely, providing more frequent feedback to adolescents and incorporating strategies (e.g. school involvement) that facilitate adolescents' proximity to their electronic devices when recordings are required.

The second aim of the study involved examining the concordance of self-report and physiologic dimensions of state affect by testing relationships between self-reported mood states and heart rate variability. Significant findings and patterns at the trait, state, and momentary levels suggested modest associations between negative, but not positive, affect and physiologic indices in which adolescents with greater negative affect exhibit the expected physiologic pattern of enhanced sympathetic and blunted parasympathetic

activity. Although these analyses were not significantly powered and require replication within an adequately powered design, they are consistent with adult and adolescent research, including those conducted in an ambulatory setting, suggesting associations between mood states and autonomic regulation.

Finally, the interpersonal model of LOC was tested, and interpersonal problems were a robust predictor of LOC eating. Although data provided preliminary support for the relationships links between interpersonal problems and negative affect, and negative affect and LOC, the mediation model was not significant. Similar to the above analyses, results should be considered in light of the limited power to examine a mediation model. Data also support potential links between physiologic and interpersonal model variables, as evidence by relationships between heart rate and heart rate variability and interpersonal problems and LOC eating. In particular, LOC episodes appear to be preceded by heart rate variability trajectories that are consistent with an autonomic stress response. Although limited by inadequate sample size, results provide preliminary support for the hypothesis that LOC episodes are preceded by aberrations in physiologic regulation.

Table 1. Participant demographics

	Completed all procedures (n = 30)		Completed baseline screening only (n = 14)		<i>p</i>
	<i>M</i>	<i>SD</i>	<i>M</i>	<i>SD</i>	
Age (y)	14.92	1.54	14.57	1.82	0.51
Race (% Non-Hispanic White)	23.3		21.4		0.89
BMI (kg/m <sup>2</sup> )	36.13	7.49	33.91	4.60	0.24
BMI-Z score <sup>a</sup>	2.19	0.43	2.14	0.39	0.73
Fat Mass (%)	39.41	7.50	38.88	5.41	0.82
Fat Free Mass (kg)	57.35	8.59	53.64	6.12	0.17

<sup>a</sup>BMI-Z score, BMI accounting for age and sex according to Centers for Disease Control and Prevention (120)

Table 2. Reliability analyses

(n = 30, k = 2029)

<i>Composite/Item</i>	<i>Alpha/M</i>	<i>N/SD</i>
<b>Interpersonal</b>	<b>0.84</b>	<b>5</b>
Argued with someone	1.77	1.17
Felt rejected	1.54	1.03
Felt lonely	1.64	1.15
Wished more friends	1.88	1.28
Wished relationships better	1.72	1.21
<b>Negative affect</b>	<b>0.89</b>	<b>6</b>
Upset	1.46	0.99
Scared	1.24	0.74
Miserable	1.42	1.03
Lonely	1.46	1.21
Blue	1.44	1.01
Sad	1.42	0.98
<b>Positive affect</b>	<b>0.93</b>	<b>6</b>
Happy	3.42	1.31
Cheerful	2.62	1.49
Proud	2.16	1.53
Joyful	2.64	1.47
Delighted	2.44	1.46
Lively	2.75	1.48

<b>Loss of Control Eating</b>	<b>0.89</b>	<b>3</b>
Sense of LOC	1.71	1.09
Lose control	1.75	1.09
Out of control at any point	1.74	1.08

Table 3. Compliance

(n = 29)

<i>Overall</i>					
<i>Rating Type</i>	<i>N</i>	<i>M</i>	<i>SD</i>	<i>Min</i>	<i>Max</i>
Random	1069	2.76			
Compliance (%)		70.00			
Before	500	1.29			
After	460	1.19			
<i>Per Subject</i>					
<i>Rating Type</i>	<i>N</i>	<i>M</i>	<i>SD</i>	<i>Min</i>	<i>Max</i>
Days	387	13.34	2.65	7	16
Random	1069	2.75	0.61	1.47	4.50
Compliant Random	897	2.30	0.52	1.25	3.00
Compliance (%)		69.36	13.62	38.00	90.00
Before	500	1.27	0.69	0.25	2.73
After	460	1.17	0.62	0.25	2.27



Table 4. Acceptability

(n = 30)

<i>Dimension</i>	<i>M</i>	<i>SD</i>	<i>Min</i>	<i>Max</i>
Overall	5.30	0.83	3.0	6.8
Easy	5.43	1.38	1.0	7.0
Disruptive	3.13	1.81	1.0	7.0
Time consuming	2.53	1.41	1.0	5.0
Helpful	4.87	1.46	1.0	7.0
Positive	5.87	1.07	3.0	7.0

Table 5. Regression analyses for prediction of compliance  
(n = 29)

<b>Interpersonal Problems</b>			
<i>Variable</i>	B	SE B	<i>p</i>
Constant	0.238	0.622	0.71
Step 1			
Race	0.065	0.057	0.26
Age	- 0.011	0.017	0.54
Step 2			
Height	0.388	0.374	0.31
Percent adiposity	0.003	0.003	0.40
Step 3			
Interpersonal Problems	- 0.085	0.035	0.02
<b>High-threshold LOC</b>			
<i>Variable</i>	B	SE B	<i>p</i>
Constant	0.437	0.565	0.45
Step 1			
Race	0.037	0.050	0.46
Age	0.003	0.016	0.84
Step 2			
Height	0.082	0.358	0.82
Percent adiposity	0.003	0.003	0.26
Step 3			
High-threshold LOC	- 0.428	0.121	< 0.01

Table 6. Heart rate and heart rate variability

(n = 19)

	<i>M</i>	<i>SD</i>
BPM <sup>a</sup>	85.48	8.85
SDNN (msec) <sup>b</sup>	74.69	18.96
Normalized LF power (nu)	0.56	0.08
Normalized HF power (nu)	0.44	0.08

<sup>a</sup>BPM = beats per minute; <sup>b</sup>SDNN (msec) = standard deviation of successive NN intervals, measured in milliseconds; nu = normalized units

Table 7. Aggregate interpersonal model variables by sub-threshold BED status

	<i>Non-BED</i>		<i>Sub-threshold BED</i>		
	<i>(n=22)</i>		<i>(n=7)</i>		
	<i>M</i>	<i>SD</i>	<i>M</i>	<i>SD</i>	<i>p</i>
Interpersonal Problems	1.74	0.74	1.90	0.89	0.65
Negative Affect	1.40	0.57	1.74	0.90	0.24
Positive Affect	2.73	1.01	2.36	0.71	0.38
Boredom	3.04	1.10	2.80	1.39	0.64
LOC	1.72	0.54	2.33	0.47	0.01
LOC high-threshold (%)	16.3	21.1	21.1	23.4	0.42

Table 8. Estimates of fixed effects from multilevel models of the relationships between momentary negative affect and heart rate, <sup>a</sup>SDNN, <sup>b</sup>RMSSD, <sup>c</sup>pNN50 and <sup>d</sup>HF/HF+LF power, examined in main effects models, split file models, and interaction models

(n = 17, k = 128)

<i>Heart Rate</i>				
<i>Variable</i>	<i>Estimate</i>	<i>SE</i>	<i>t</i>	<i>p</i>
<i>Main Effects Model</i>				
Intercept	81.10	2.68	30.23	< 0.001
Negative Affect (log)	22.21	6.55	3.39	< 0.01
Posture	11.11	2.51	4.43	< 0.001
<i>Split file—Random recordings</i>				
Intercept	79.51	3.17	25.10	< 0.001
Negative affect (log)	21.01	8.44	2.49	0.02
Posture	11.13	2.83	3.93	< 0.001
<i>Split file—Before-meal</i>				
Intercept	86.60	6.29	13.77	< 0.001
Negative affect (log)	29.12	12.99	2.42	0.04
Posture	9.29	6.62	1.41	0.17
<i>Split file—After-meal</i>				
Intercept	88.17	5.57	15.84	< 0.001
Negative affect (log)	7.93	10.39	0.76	0.45
Posture	5.77	5.66	1.02	0.32
<i>Interaction Model</i>				
Intercept	83.53	3.52	23.72	< 0.001

Posture	10.15	2.46	4.12	< 0.001
Ratatype (random)	-3.35	2.81	-1.19	0.24
Ratatype (before)	2.89	3.74	0.77	0.44
Ratatype (after)	0	0	-	-
Negative affect (log)	13.14	10.17	1.29	0.20
Ratatype (random) * Negative affect	7.01	12.05	0.58	0.56
Ratatype (before) * Negative affect	15.99	14.48	1.10	0.27
Ratatype (after) * Negative affect	0	0	-	-

<sup>a</sup>*SDNN*

<i>Variable</i>	<i>Estimate</i>	<i>SE</i>	<i>t</i>	<i>p</i>
<i>Main Effects Model</i>				
Intercept	80.19	8.02	9.99	< 0.001
Negative Affect (log)	-9.14	18.98	-0.48	0.63
Posture	-9.08	7.03	-1.29	0.20
<i>Split file—Random recordings</i>				
Intercept	78.10	10.41	7.51	< 0.001
Negative affect (log)	6.64	26.12	0.25	0.80
Posture	-2.41	8.45	-0.29	0.78
<i>Split file—Before-meal</i>				
Intercept	72.85	15.92	4.58	< 0.001
Negative affect (log)	-11.33	34.03	-0.33	0.74
Posture	-7.15	16.32	-0.44	0.67



<i>Split file—After-meal</i>				
Intercept	75.95	14.91	5.09	< 0.001
Negative affect (log)	-4.99	26.37	-0.19	0.85
Posture	-14.20	15.57	-0.91	0.37
<i>Interaction Model</i>				
Intercept	72.66	10.37	7.01	< 0.001
Posture	-6.75	7.11	-0.95	0.34
Ratatype (random)	7.47	8.04	0.93	0.35
Ratatype (before)	1.96	10.68	0.18	0.86
Ratatype (after)	0	0	-	-
Negative affect (log)	-21.22	29.41	-0.72	0.47
Ratatype (random) * Negative affect	31.35	34.46	0.91	0.37
Ratatype (before) * Negative affect	5.90	41.38	0.14	0.89
Ratatype (before) * Negative affect	0	0	-	-
<sup>b</sup> <i>RMSSD</i>				
<i>Variable</i>	<i>Estimate</i>	<i>SE</i>	<i>t</i>	<i>p</i>
<i>Main Effects Model</i>				
Intercept	64.46	6.97	9.25	< 0.001
Negative Affect (log)	-13.92	17.34	-0.80	0.43
Posture	-17.95	6.89	-2.61	0.01
<i>Split file—Random recordings</i>				
Intercept	67.87	9.95	6.82	< 0.001

Negative affect (log)	-13.07	27.34	-0.48	0.63
Posture	-13.88	9.36	-1.48	0.14
<i>Split file—Before-meal</i>				
Intercept	48.49	10.69	4.53	< 0.001
Negative affect (log)	3.98	22.10	0.18	0.86
Posture	-13.53	11.25	-1.20	0.24
<i>Split file—After-meal</i>				
Intercept	56.31	11.23	5.02	< 0.001
Negative affect (log)	-2.64	19.86	-0.13	0.90
Posture	-17.73	11.73	-1.51	0.14
<i>Interaction Model</i>				
Intercept	54.04	9.68	5.58	< 0.001
Posture	-14.26	6.86	-2.08	0.04
Ratatype (random)	12.95	7.91	1.64	0.11
Ratatype (before)	-5.17	10.51	-0.49	0.62
Ratatype (after)	0	0	-	-
Negative affect (log)	-15.59	28.31	-0.55	0.58
Ratatype (random) * Negative affect	10.77	33.86	0.32	0.75
Ratatype (before) * Negative affect	8.32	40.71	0.20	0.84
Ratatype (after) * Negative affect	0	0	-	-
<sup>c</sup> <i>pNN50</i>				
<i>Variable</i>	<i>Estimate</i>	<i>SE</i>	<i>t</i>	<i>p</i>

<i>Main Effects Model</i>				
Intercept	15.30	1.74	8.77	< 0.001
Negative Affect (log)	-6.67	4.26	-1.56	0.12
Posture	-6.49	1.64	-3.96	< 0.001
<i>Split file—Random recordings</i>				
Intercept	15.87	2.33	6.83	< 0.001
Negative affect (log)	-5.73	6.09	-0.94	0.35
Posture	-5.43	2.02	-2.69	< 0.01
<i>Split file—Before-meal</i>				
Intercept	11.75	3.06	3.85	0.001
Negative affect (log)	-6.10	6.31	-0.97	0.34
Posture	-4.45	3.21	-1.38	0.18
<i>Split file—After-meal</i>				
Intercept	14.03	3.29	4.26	< 0.001
Negative affect (log)	-2.28	5.82	-0.39	0.70
Posture	-6.82	3.44	-1.98	0.06
<i>Interaction Model</i>				
Intercept	13.03	2.34	5.56	< 0.001
Posture	-5.49	1.62	-3.38	0.001
Ratatype (random)	2.51	1.84	1.36	0.18
Ratatype (before)	-1.18	2.45	-0.48	0.63
Ratatype (after)	0	0	-	-
Negative affect (log)	-7.91	6.70	-1.18	0.24
Ratatype (random) * Negative	5.27	7.89	0.67	0.50

affect				
Ratetype (before) * Negative affect	-0.34	9.47	-0.04	0.97
affect				
Ratetype (before) * Negative affect	0	0	-	-
<sup>d</sup> <i>HF/HF+LF</i>				
<i>Variable</i>	<i>Estimate</i>	<i>SE</i>	<i>t</i>	<i>p</i>
<i>Main Effects Model</i>				
Intercept	0.441	0.040	11.13	< 0.001
Negative Affect (log)	-0.004	0.098	-0.04	0.97
Posture	-0.047	0.038	-1.23	0.22
<i>Split file—Random recordings</i>				
Intercept	0.434	0.044	9.89	< 0.001
Negative affect (log)	0.091	0.124	0.73	0.47
Posture	-0.033	0.043	-0.76	0.45
<i>Split file—Before-meal</i>				
Intercept	0.402	0.111	3.61	< 0.01
Negative affect (log)	0.107	0.230	0.46	0.64
Posture	-0.064	0.117	-0.54	0.59
<i>Split file—After-meal</i>				
Intercept	0.494	0.082	6.02	< 0.001
Negative affect (log)	-0.132	0.154	-0.86	0.40
Posture	0.079	0.083	-0.95	0.35
<i>Interaction Model</i>				

Intercept	0.478	0.054	8.90	< 0.001
Posture	-0.038	0.038	-1.00	0.32
Ratetype (random)	-0.047	0.044	-1.07	0.29
Ratetype (before)	-0.101	0.058	-1.74	0.09
Ratetype (after)	0	0	-	-
Negative affect (log)	-0.234	0.157	-1.49	0.14
Ratetype (random) * Negative affect	0.362	0.188	1.92	0.06
Ratetype (before) * Negative affect	0.324	0.226	1.43	0.16
Ratetype (before) * Negative affect	0	0	-	-

<sup>a</sup>SDNN (msec) = standard deviation of successive NN intervals, measured in milliseconds; <sup>b</sup>RMSSD (msec) = root mean square of successive differences in beat-to-beat intervals, measured in milliseconds; <sup>c</sup>pNN50 = proportion of beat-to-beat intervals differing by greater than 50 milliseconds; <sup>d</sup>HF/HF+LF = high frequency power divided by total power

Table 9. Multilevel models of heart rate, <sup>a</sup>SDNN, <sup>b</sup>RMSSD, <sup>c</sup>pNN50, and <sup>d</sup>HF/HF+LF over time following high and low interpersonal problem ratings

(n = 14, k = 48)

<i>Heart Rate</i>				
<i>Variable</i>	<i>Estimate</i>	<i>SE</i>	<i>t</i>	<i>p</i>
Intercept	89.60	3.32	26.96	< 0.001
Interpersonal P	13.78	2.96	4.66	< 0.001
Time (hrs)	-7.41	4.12	-1.80	0.07
Interpersonal P * Time (hrs)	-1.60	7.39	-0.22	0.83
Time(hrs) <sup>2</sup>	3.16	3.30	0.96	0.34
Interpersonal P * Time (hrs) <sup>2</sup>	-1.48	5.86	-0.25	0.80
Time(hrs) <sup>3</sup>	-0.51	0.74	-0.69	0.49
Interpersonal P * Time (hrs) <sup>3</sup>	0.74	1.31	0.56	0.57
<sup>a</sup> SDNN				
<i>Variable</i>				
Intercept	84.02	8.87	9.47	< 0.001
Interpersonal P	-4.11	7.13	-0.58	0.56
Time (hrs)	-28.44	9.89	-2.88	< 0.01
Interpersonal P * Time (hrs)	19.32	17.75	1.09	0.28
Time(hrs) <sup>2</sup>	15.73	7.93	1.98	0.05
Interpersonal P * Time (hrs) <sup>2</sup>	-9.70	14.07	-0.69	0.49
Time(hrs) <sup>3</sup>	-2.15	1.78	-1.21	0.23
Interpersonal P * Time (hrs) <sup>3</sup>	1.26	3.14	0.40	0.69
<sup>b</sup> RMSSD				



<i>Variable</i>				
Intercept	57.24	9.59	5.97	< 0.001
Interpersonal P	-14.74	7.17	-2.06	0.04
Time (hrs)	0.99	9.94	0.10	0.92
Interpersonal P * Time (hrs)	15.78	17.84	0.88	0.38
Time(hrs) <sup>2</sup>	1.65	7.97	0.21	0.84
Interpersonal P * Time (hrs) <sup>2</sup>	-13.40	14.14	-0.95	0.34
Time(hrs) <sup>3</sup>	-0.33	1.79	-0.19	0.85
Interpersonal P * Time (hrs) <sup>3</sup>	3.08	3.15	0.98	0.33

<sup>c</sup>*pNN50*

<i>Variable</i>				
Intercept	11.93	2.16	5.53	< 0.001
Interpersonal P	-6.33	1.93	-3.29	0.001
Time (hrs)	4.73	2.68	1.77	0.08
Interpersonal P * Time (hrs)	-1.40	4.81	-0.29	0.77
Time(hrs) <sup>2</sup>	-3.26	2.15	-1.52	0.13
Interpersonal P * Time (hrs) <sup>2</sup>	1.71	3.81	0.45	0.65
Time(hrs) <sup>3</sup>	0.67	0.48	1.39	0.17
Interpersonal P * Time (hrs) <sup>3</sup>	-0.45	0.85	-0.53	0.60

<sup>d</sup>*HF/HF+LF*

<i>Variable</i>				
Intercept	0.36	0.03	11.48	< 0.001
Interpersonal P	- 0.01	0.04	-0.34	0.73
Time (hrs)	0.25	0.05	4.65	< 0.001

Interpersonal P * Time (hrs)	-0.28	0.10	-2.91	< 0.01
Time(hrs) <sup>2</sup>	-0.14	0.04	-3.29	0.001
Interpersonal P * Time (hrs) <sup>2</sup>	0.18	0.08	2.34	0.02
Time(hrs) <sup>3</sup>	0.02	0.01	2.49	0.01
Interpersonal P * Time (hrs) <sup>3</sup>	-0.03	0.02	-1.97	0.05

Interpersonal P = Interpersonal Problems; <sup>a</sup>SDNN (msec) = standard deviation of successive NN intervals, measured in milliseconds; <sup>b</sup>RMSSD (msec) = root mean square of successive differences in beat-to-beat intervals, measured in milliseconds; <sup>c</sup>pNN50 = proportion of beat-to-beat intervals differing by greater than 50 milliseconds; <sup>d</sup>HF/HF+LF = high frequency power divided by total power

Table 10. Multilevel models of heart rate, <sup>a</sup>SDNN, <sup>b</sup>RMSSD, <sup>c</sup>pNN50, and <sup>d</sup>HF/HF+LF over time prior to LOC and non-LOC eating episodes

(n = 17, k = 43)

<i>Heart Rate</i>				
<i>Variable</i>	<i>Estimate</i>	<i>SE</i>	<i>t</i>	<i>p</i>
Intercept	92.37	6.60	14.00	0.00
LOC	38.49	18.99	2.03	0.04
Time (hrs)	13.14	12.49	1.05	0.29
LOC * Time (hrs)	71.37	39.65	1.80	0.07
Time(hrs) <sup>2</sup>	10.41	7.80	1.34	0.18
LOC * Time (hrs) <sup>2</sup>	54.61	24.79	2.20	0.03
Time(hrs) <sup>3</sup>	2.29	1.49	1.54	0.12
LOC * Time (hrs) <sup>3</sup>	11.52	4.74	2.43	0.02
<sup>a</sup> SDNN				
<i>Variable</i>				
Intercept	75.91	14.45	5.25	0.00
LOC	-79.42	38.07	-2.09	0.04
Time (hrs)	-17.11	25.02	-0.68	0.49
LOC * Time (hrs)	-143.58	79.45	-1.81	0.07
Time(hrs) <sup>2</sup>	-10.24	15.62	-0.66	0.51
LOC * Time (hrs) <sup>2</sup>	-105.51	49.67	-2.12	0.03
Time(hrs) <sup>3</sup>	-1.80	2.98	-0.61	0.55
LOC * Time (hrs) <sup>3</sup>	-22.24	9.49	-2.34	0.02
<sup>b</sup> RMSSD				

<i>Variable</i>				
Intercept	45.17	14.56	3.10	< 0.01
LOC	-127.03	35.96	-3.53	< 0.001
Time (hrs)	-39.04	23.64	-1.65	0.10
LOC * Time (hrs)	-254.70	75.05	-3.39	< 0.01
Time(hrs) <sup>2</sup>	-24.90	14.76	-1.69	0.09
LOC * Time (hrs) <sup>2</sup>	-185.33	46.92	-3.95	< 0.001
Time(hrs) <sup>3</sup>	-4.77	2.82	-1.69	0.09
LOC * Time (hrs) <sup>3</sup>	-38.59	8.97	-4.30	< 0.001

<sup>c</sup>*pNN50*

<i>Variable</i>				
Intercept	10.27	3.83	2.68	< 0.01
LOC	-33.02	10.82	-3.05	< 0.01
Time (hrs)	-6.77	7.11	-0.95	0.34
LOC * Time (hrs)	-66.21	22.59	-2.93	< 0.01
Time(hrs) <sup>2</sup>	-4.82	4.44	-1.09	0.28
LOC * Time (hrs) <sup>2</sup>	-48.48	14.12	-3.43	< 0.01
Time(hrs) <sup>3</sup>	-1.01	0.85	-1.19	0.24
LOC * Time (hrs) <sup>3</sup>	-10.11	2.70	-3.75	< 0.001

<sup>d</sup>*HF/HF+LF*

<i>Variable</i>				
Intercept	0.29	0.06	4.60	< 0.001
LOC	- 0.25	0.19	-1.31	0.19
Time (hrs)	- 0.30	0.13	-2.38	0.02

LOC * Time (hrs)	- 0.38	0.40	-0.94	0.35
Time(hrs) <sup>2</sup>	- 0.21	0.08	-2.60	< 0.01
LOC * Time (hrs) <sup>2</sup>	- 0.26	0.25	-1.03	0.30
Time(hrs) <sup>3</sup>	- 0.04	0.02	-2.74	< 0.01
LOC * Time (hrs) <sup>3</sup>	- 0.05	0.05	-1.13	0.26

LOC = Loss of Control eating; <sup>a</sup>SDNN (msec) = standard deviation of successive NN intervals, measured in milliseconds; <sup>b</sup>RMSSD (msec) = root mean square of successive differences in beat-to-beat intervals, measured in milliseconds; <sup>c</sup>pNN50 = proportion of beat-to-beat intervals differing by greater than 50 milliseconds; <sup>d</sup>HF/HF+LF = high frequency power divided by total power

Figure 1. Interpersonal model of Loss of Control (LOC) eating, positing that interpersonal problems precede and predict negative affect, which in turn precedes and predicts LOC eating. Negative affect is thought to serve as a mediating variable between interpersonal problems and LOC eating.

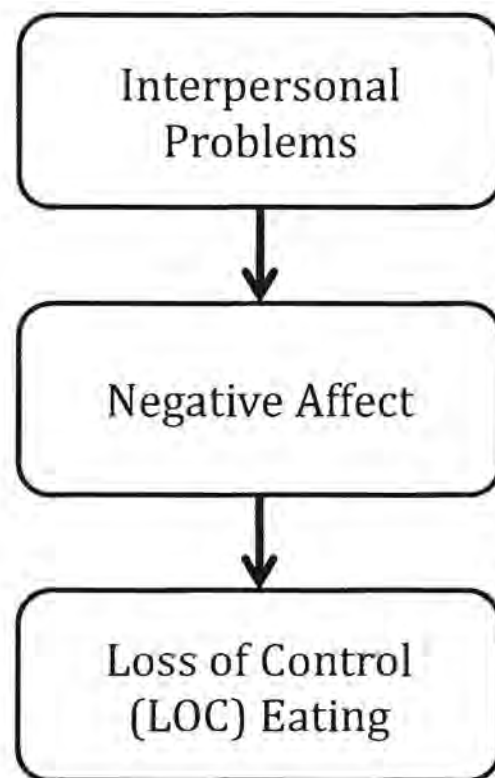




Figure 2. Model of negative affect as a mediator of social problems and LOC eating, controlling for age, sex, race, pubertal status, and BMI-Z score.

\* $p < 0.05$ , \*\* $p < 0.01$ , \*\*\* $p < 0.001$ . Reprinted from "An examination of the interpersonal model of loss of control eating in children and adolescents" by C. A. Elliott, M. Tanofsky-Kraff, L. B. Shomaker, K. M. Columbo, L. E. Wolkoff, L. M. Ranzenhofer, and J. A. Yanovski, 2010, *Behavior Research and Therapy*, 48(5), p. 424-428.

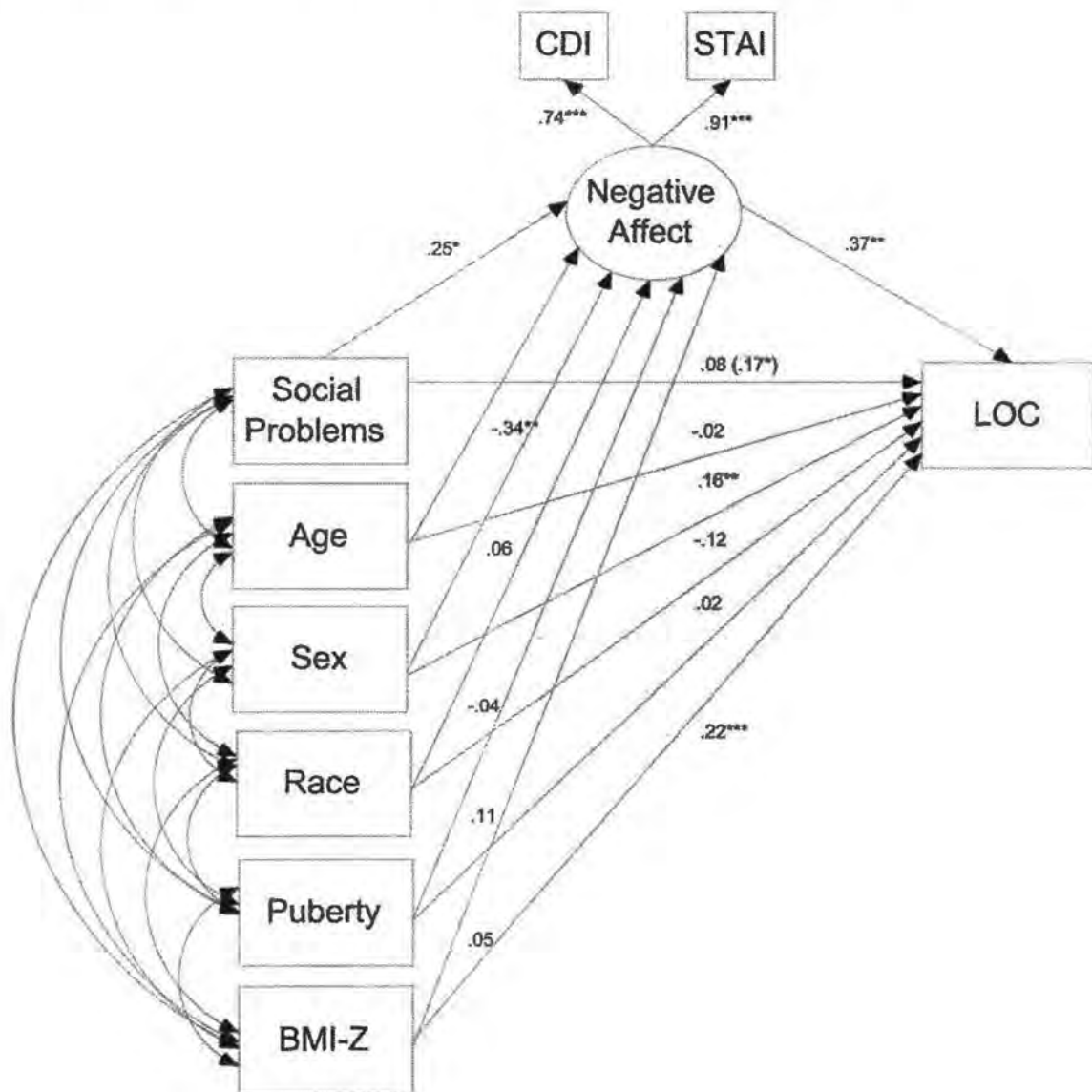


Figure 3. Steps involved in testing mediation. Path c: demonstrate that the initial variable (X) is correlated with the outcome variable (Y). Path a: demonstrate that the initial variable (X) is correlated with the mediator variable (M). Path b: while controlling for the initial variable, show that the mediator variable (M) is correlated with the outcome variable (Y). Path c': to establish the degree to which M is a mediator between X and Y, calculate the degree to which the correlation between X and Y is reduced when controlling for M.

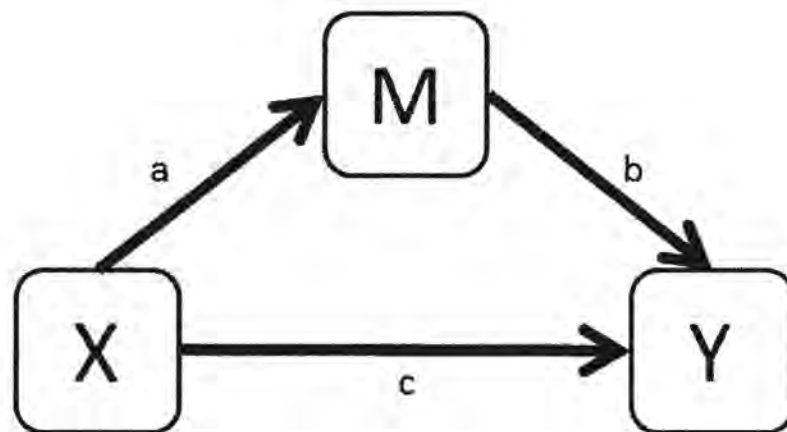


Figure 4. Depression and anxiety scores on the Beck Depression Inventory-II (BDI-II) and the State-Trait Anxiety Inventory-Child, trait version, for sub-threshold BED and non-BED groups

(n = 30)

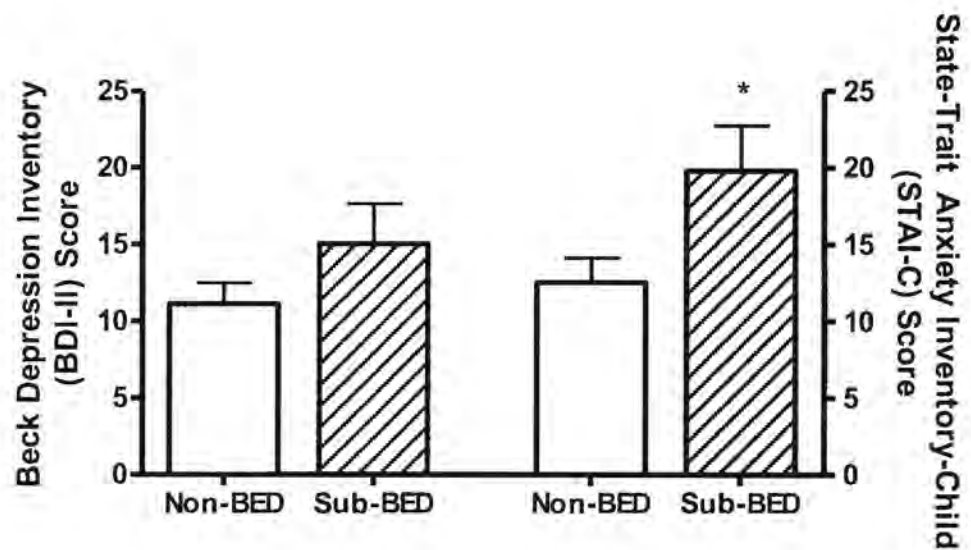


Figure 5. Total, internalizing, and externalizing psychiatric symptoms as measured by the Kiddie Schedule for Affective Disorders and Schizophrenia (KSADS) for sub-threshold BED and non-BED groups

(n = 30)

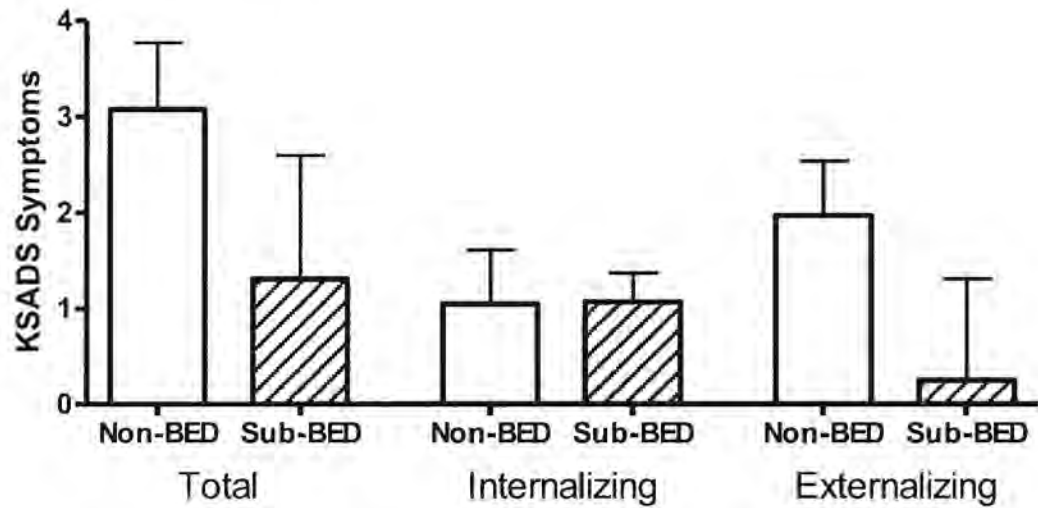


Figure 6. Reasons for non-compliance reported by adolescents

(n = 30)

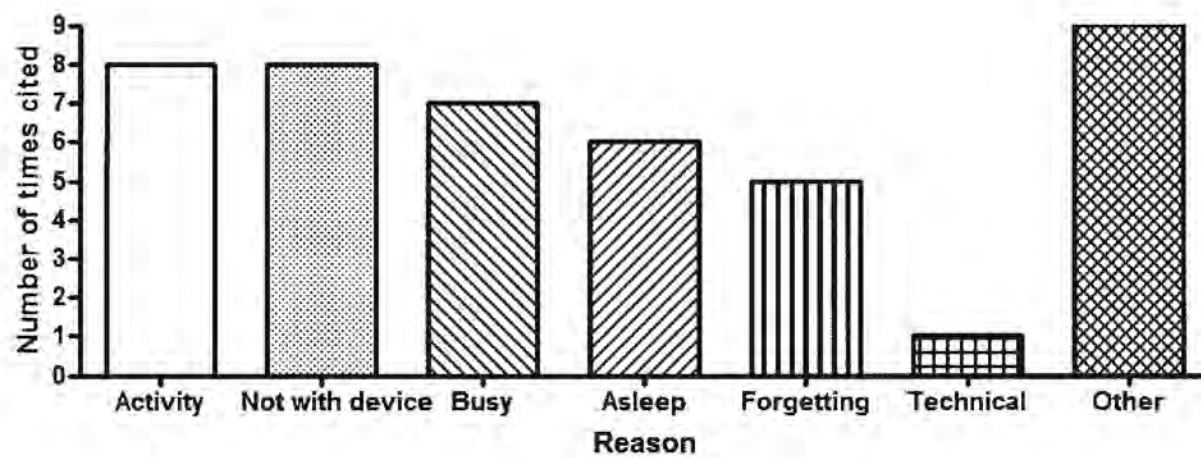


Figure 7. Relationships between daily normalized HF power and total psychiatric symptoms and internalizing symptoms

( $n = 18, k = 33$ )

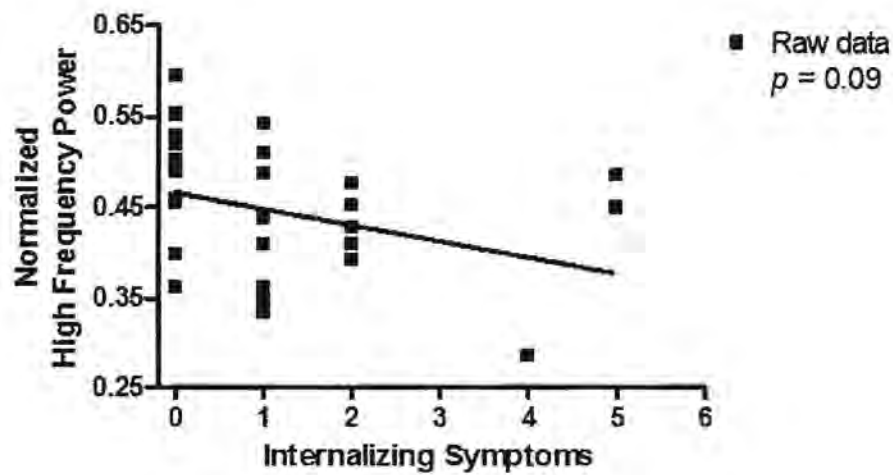
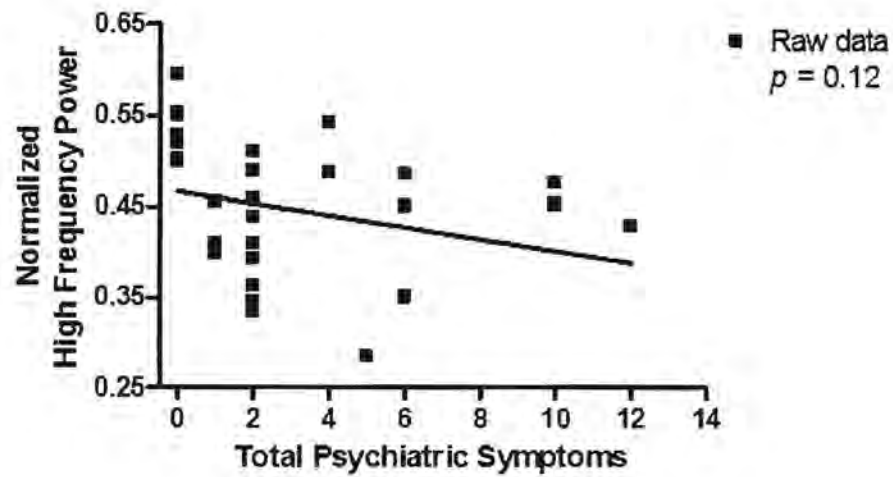




Figure 8. Relationships between daily normalized HF power and dimensions of state affect including negative affect, positive affect, and boredom

( $n = 17$ ,  $k = 30$ )

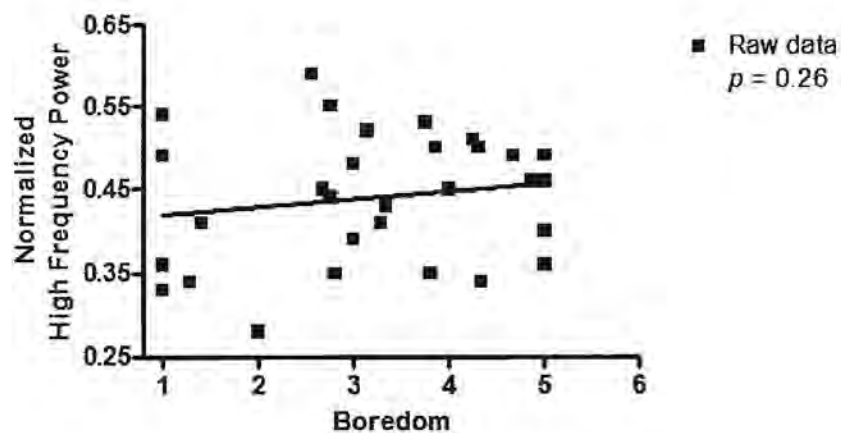
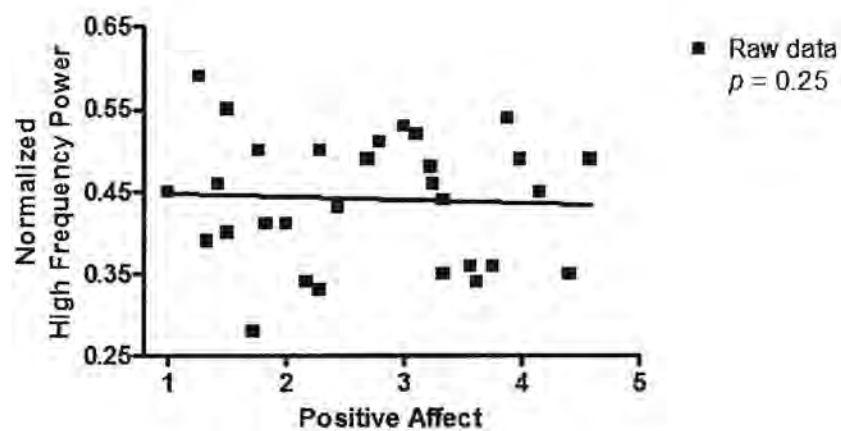
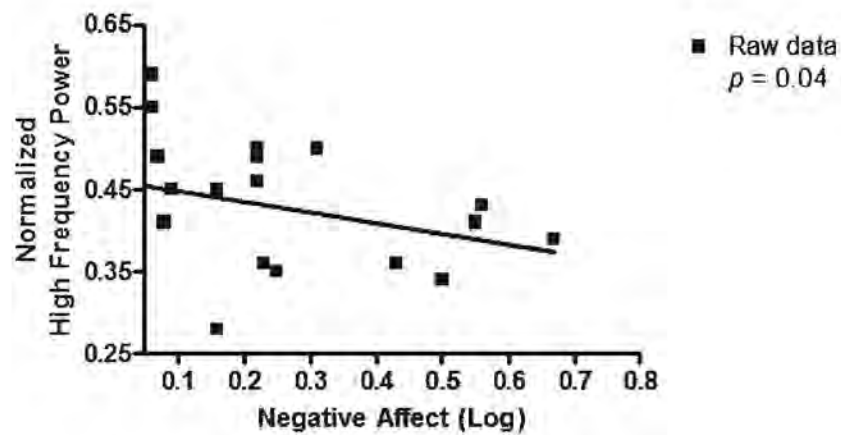


Figure 9. Relationship between momentary negative affect (log transformed) and heart rate for random, before-meal, and after-meal recordings

( $n = 17$ ,  $k = 128$ )

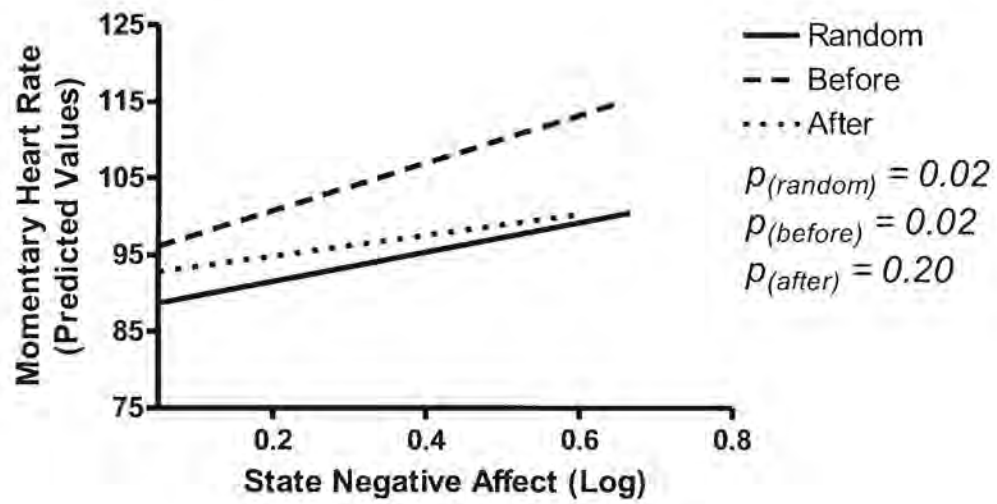


Figure 10. Relationship between momentary positive affect and the proportion of HF power for random, before-meal, and after-meal recordings

( $n = 17$ ,  $k = 128$ )

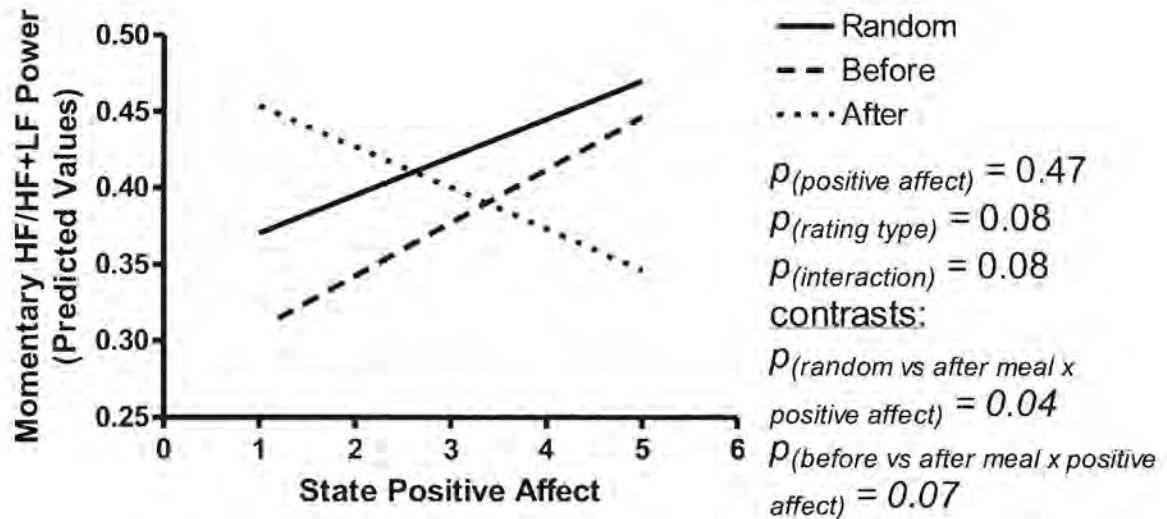


Figure 11. Interpersonal model: path c. Relationship between interpersonal problems (X) at time 1 and LOC eating (Y) at time 2. In this analysis, interpersonal problems directly preceded LOC ratings, with measurements occurring approximately one half hour apart (n = 30, k = 426).

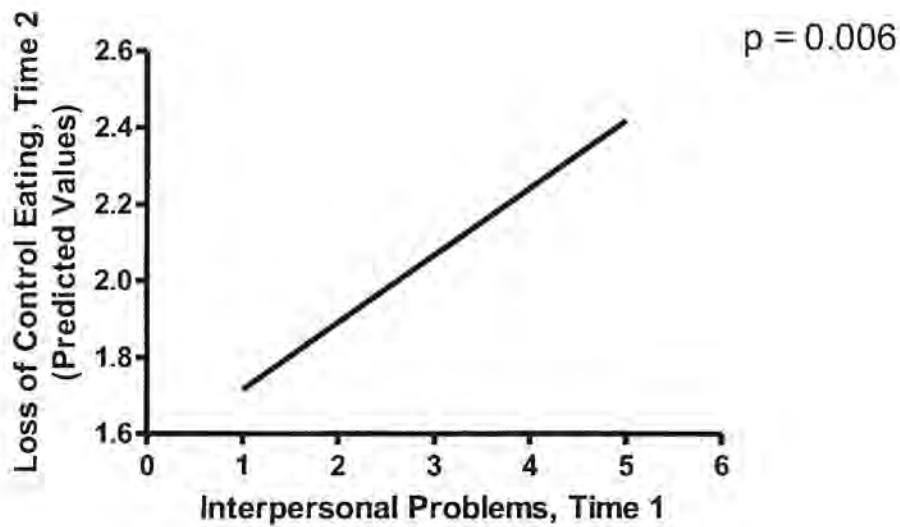


Figure 12. Interpersonal model: path a: Relationship between interpersonal problems (X) at time 1 and state affect (M) at time 2, with measurements occurring approximately one half hour apart. Shown are negative affect, positive affect, and boredom.

( $n = 29$ ,  $k = 641$ )

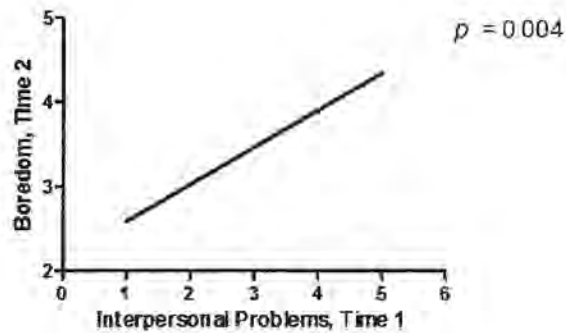
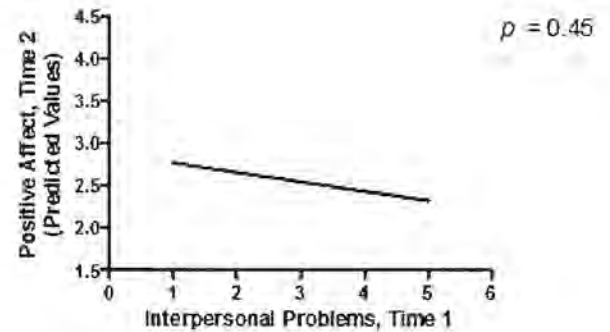
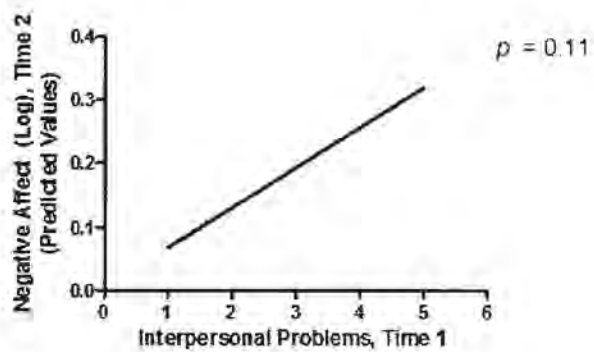


Figure 13. Interpersonal model: path b: Relationship between negative affect (M) and LOC Eating (Y). The first graph depicts the relationship between negative affect at time 1 and LOC Eating at time 2 ( $n = 30$ ,  $k = 426$ ). The second graph depicts the relationship between negative affect at time 2, controlling for negative affect at time 1 (thereby representing the change in negative affect) and LOC Eating at time 3 ( $n = 29$ ,  $k = 184$ ).

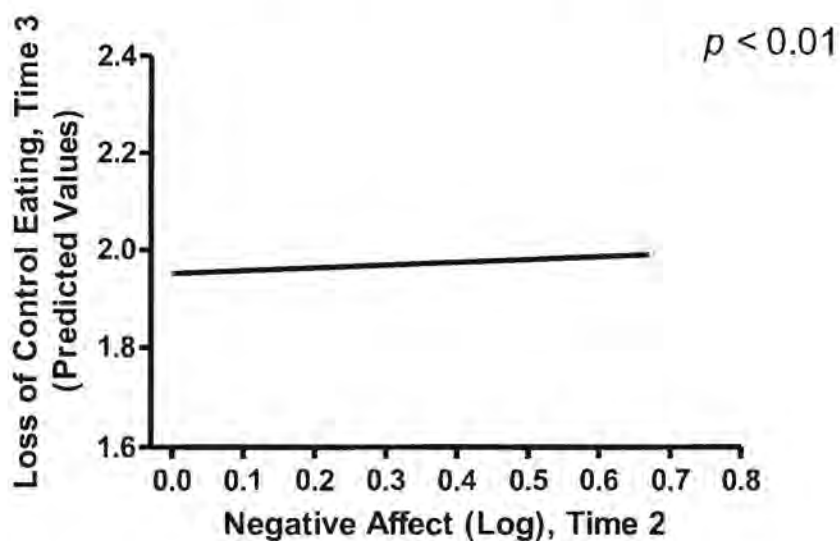
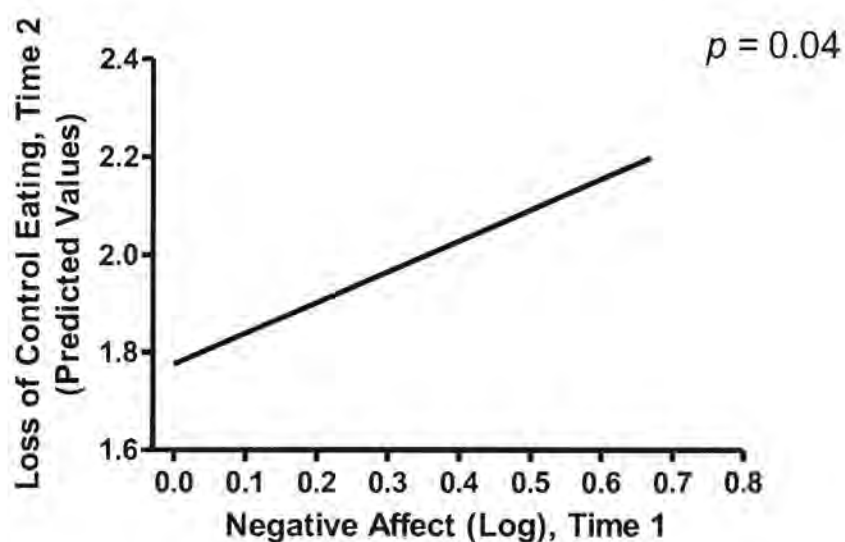




Figure 14. Trajectories of heart rate, SDNN, RMSSD, pNN50, and HF-HRV during the 3 hours preceding after-meal ratings for “high” and “low” LOC episodes

(n = 17, k = 43)

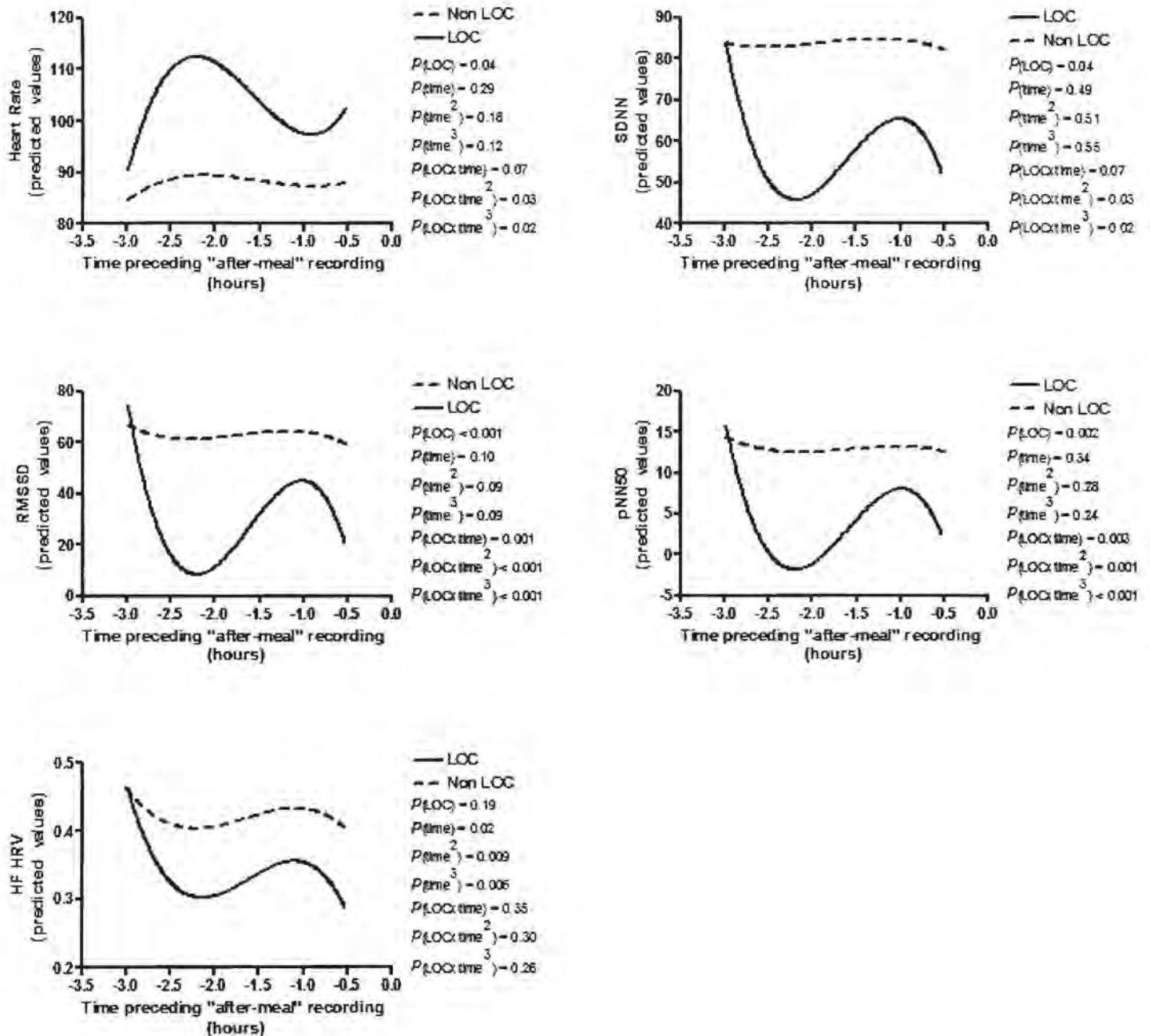
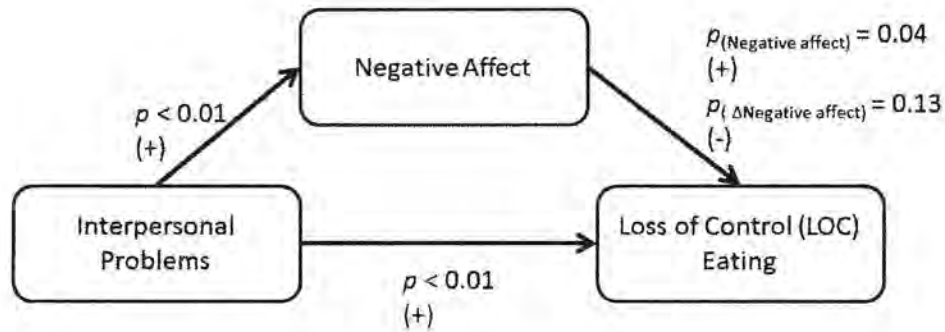


Figure 15. Summary of findings from aim 3 hypotheses 1 – 3 for negative affect, using  
(a) all cases and (b) only cases included in the mediation model

a.  $n = 30$ ,  $k_{\text{path a}} = 641$ ,  $k_{\text{path b,c}} = 426$



b.  $n = 29$ ,  $k = 184$

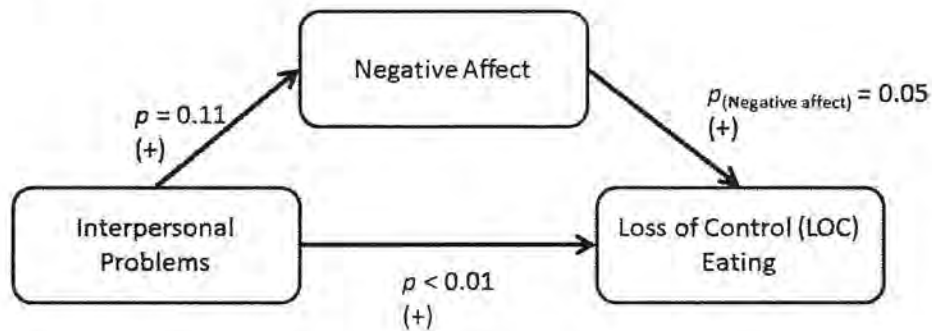
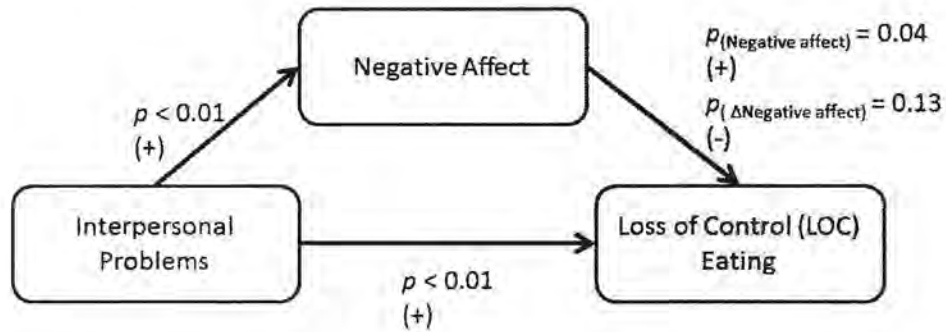


Figure 16. Summary of findings from aim 3 hypotheses 1 – 3 for boredom, using (a) all cases and (b) only cases included in the mediation model.

a.  $n = 30$ ,  $k_{\text{path a}} = 641$ ,  $k_{\text{path b,c}} = 426$



b.  $n = 29$ ,  $k = 184$

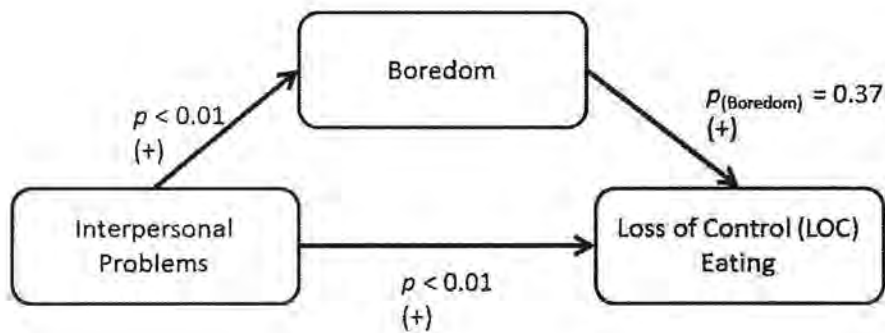
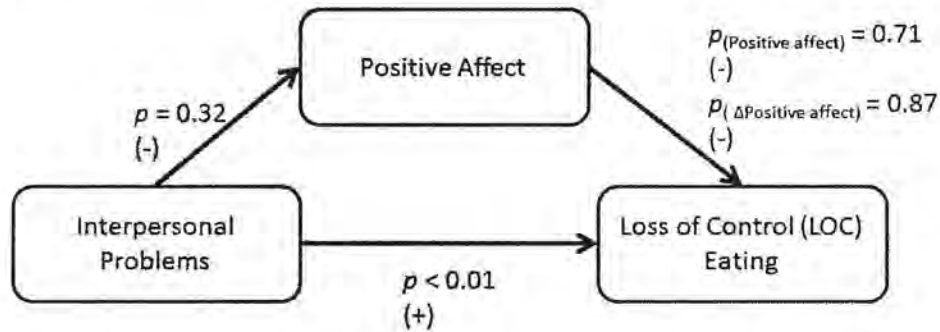
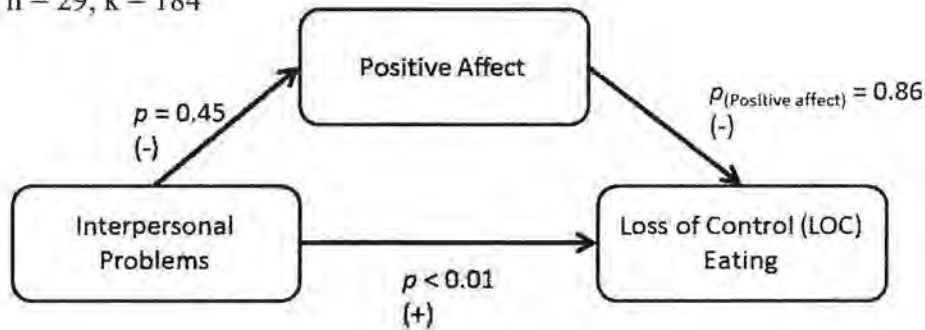


Figure 17. Summary of findings from aim 3 hypotheses 1 – 3 for positive affect, using (a) all cases and (b) only cases included in the mediation model.

a.  $n = 30$ ,  $k_{\text{path a}} = 641$ ,  $k_{\text{path b,c}} = 426$



b.  $n = 29$ ,  $k = 184$



## REFERENCES

1. Adam EK. 2006. Transactions among adolescent trait and state emotion and diurnal and momentary cortisol activity in naturalistic settings. *Psychoneuroendocrinology* 31:664-79
2. Agelink MW, Boz C, Ullrich H, Andrich J. 2002. Relationship between major depression and heart rate variability. Clinical consequences and implications for antidepressive treatment. *Psychiatry Res* 113:139-49
3. Agras WS, Telch CF. 1998. The effects of caloric deprivation and negative affect on binge eating in binge-eating disordered women. *Behavior Therapy* 29:491-503
4. Alegria M, Woo M, Cao Z, Torres M, Meng XL, Striegel-Moore R. 2007. Prevalence and correlates of eating disorders in Latinos in the United States. *Int J Eat Disord* 40 Suppl:S15-21
5. Allen JP, Hauser ST, Bell KL, O'Connor TG. 1994. Longitudinal assessment of autonomy and relatedness in adolescent-family interactions as predictors of adolescent ego development and self-esteem. *Child Dev* 65:179-94
6. Allen JP, Hauser ST, Eickholt C, Bell KL, O'Connor TG. 1994. Autonomy and Relatedness in Family Interactions as Predictors of Expressions of Negative Adolescent Affect. *Journal of Research on Adolescence* 4:535-52
7. Ambarish V, Barde P, Vyas A, Deepak KK. 2005. Comparison between pre-prandial and post-prandial heart rate variability (HRV). *Indian J Physiol Pharmacol* 49:436-42

8. American Psychiatric Association. 2000. Diagnostic and Statistical Manual of Mental Disorders, 4th edition, text revision (DSM-IV-TR). Washington, DC: American Psychiatric Association
9. American Psychiatric Association. 2012. *DSM-V Development: Binge Eating Disorder*.  
<http://www.dsm5.org/ProposedRevision/Pages/proposedrevision.aspx?rid=372>
10. Anderson B, Ho J, Brackett J, Finkelstein D, Laffel L. 1997. Parental involvement in diabetes management tasks: relationships to blood glucose monitoring adherence and metabolic control in young adolescents with insulin-dependent diabetes mellitus. *The Journal of pediatrics* 130:257-65
11. Anderson VA, Anderson P, Northam E, Jacobs R, Catroppa C. 2001. Development of executive functions through late childhood and adolescence in an Australian sample. *Developmental neuropsychology* 20:385-406
12. Ansell EB, Grilo CM, White MA. 2012. Examining the interpersonal model of binge eating and loss of control over eating in women. *Int J Eat Disord* 45:43-50
13. Appelhans BM, Luecken LJ. 2006. Heart rate variability as an index of regulated emotional responding. *Journal of General Psychology* 10:229-40
14. Arnett JJ. 1999. Adolescent storm and stress, reconsidered. *Am Psychol* 54:317-26
15. Arnow B, Kenardy J, Agras WS. 1992. Binge eating among the obese: a descriptive study. *Journal of Behavioral Medicine* 15:155-70
16. Axelson DA, Bertocci MA, Lewin DS, Trubnick LS, Birmaher B, et al. 2003. Measuring mood and complex behavior in natural environments: use of ecological



- momentary assessment in pediatric affective disorders. *Journal of child and adolescent psychopharmacology* 13:253-66
17. Baron RM, Kenny DA. 1986. The moderator-mediator variable distinction in social psychological research: conceptual, strategic, and statistical considerations. *J Pers Soc Psychol* 51:1173-82
  18. Beck AT, Steer RA, Brown GK. 1996. *Manual for the Beck Depression Inventory-II*. San Antonio, TX: Psychological Corporation
  19. Behrens JT. 1997. Principles and procedures of exploratory data analysis. *Psychological Methods* 2:131-60
  20. Bennett DS, Ambrosini PJ, Bianchi M, Barnett D, Metz C, Rabinovich H. 1997. Relationship of Beck Depression Inventory factors to depression among adolescents. *J Affect Disord* 45:127-34
  21. Berg KC, Crosby RD, Cao L, Peterson CB, Engel SG, et al. 2013. Facets of negative affect prior to and following binge-only, purge-only, and binge/purge events in women with bulimia nervosa. *J Abnorm Psychol* 122:111-8
  22. Bhattacharyya MR, Whitehead DL, Rakhit R, Steptoe A. 2008. Depressed mood, positive affect, and heart rate variability in patients with suspected coronary artery disease. *Psychosom Med* 70:1020-7
  23. Bishop JA. 1995. Peer Acceptance and Friendship: An Investigation of their Relation to Self-Esteem. *Journal of Early Adolescence* 15:476-89
  24. Bolger N, Zuckerman A. 1995. A framework for studying personality in the stress process. *J Pers Soc Psychol* 69:890-902

25. Bosch JA, de Geus EJ, Carroll D, Goedhart AD, Anane LA, et al. 2009. A general enhancement of autonomic and cortisol responses during social evaluative threat. *Psychosom Med* 71:877-85
26. Bosch NM, Riese H, Ormel J, Verhulst F, Oldehinkel AJ. 2009. Stressful life events and depressive symptoms in young adolescents: Modulation by respiratory sinus arrhythmia? The TRAILS study. *Biol Psychol* 81:40-7
27. Bowlby J. 1973. *Attachment and loss: Separation*. New York: Basic Books
28. Brendgen M, Lamarche V, Wanner B, Vitaro F. 2010. Links between friendship relations and early adolescents' trajectories of depressed mood. *Dev Psychol* 46:491-501
29. Brody ML, Walsh BT, Devlin MJ. 1994. Binge eating disorder: reliability and validity of a new diagnostic category. *J Consult Clin Psychol* 62:381-6
30. Brosschot JF, Van Dijk E, Thayer JF. 2007. Daily worry is related to low heart rate variability during waking and the subsequent nocturnal sleep period. *Int J Psychophysiol* 63:39-47
31. Bruce B, Agras WS. 1992. Binge eating in females: A population-based investigation. *Int J Eat Disord* 12:365-73
32. Cachelin FM, Striegel-Moore RH, Elder KA, Pike KM, Wilfley DE, Fairburn CG. 1999. Natural course of a community sample of women with binge eating disorder. *Int J Eat Disord* 25:45-54
33. Carano A, De Berardis D, Gambi F, Di Paolo C, Campanella D, et al. 2006. Alexithymia and body image in adult outpatients with binge eating disorder. *Int J Eat Disord* 39:332-40

34. Castonguay A, Eldredge KL, Agras WS. 1995. Binge Eating Disorder: current state and future directions. *Clinical Psychology Review* 15:865-90.
35. Chua JL, Touyz S, Hill AJ. 2004. Negative mood-induced overeating in obese binge eaters: an experimental study. *Int J Obes Relat Metab Disord* 28:606-10
36. Cortina J. 1993. What is coefficient alpha? An examination of theory and applications. *Journal of Applied Psychology* 78:98-104
37. Croll J, Neumark-Sztainer D, Story M, Ireland M. 2002. Prevalence and risk and protective factors related to disordered eating behaviors among adolescents: relationship to gender and ethnicity. *Journal of Adolescent Health* 31:166-75
38. Czaja J, Hartmann AS, Rief W, Hilbert A. 2011. Mealtime family interactions in home environments of children with loss of control eating. *Appetite* 56:587-93
39. Czaja J, Rief W, Hilbert A. 2009. Emotion regulation and binge eating in children. *Int J Eat Disord* 42:356-62
40. de Zwaan M. 2001. Binge eating disorder and obesity. *Int J Obes Relat Metab Disord* 25 Suppl 1:S51-5
41. de Zwaan M, Bach M, Mitchell JE, Ackard D, Specker SM, et al. 1995. Alexithymia, obesity, and binge eating disorder. *Int J Eat Disord* 17:135-40
42. Deaver CM, Miltenberger RG, Smyth J, Meidinger A, Crosby R. 2003. An evaluation of affect and binge eating. *Behavior Modification* 27:578-99
43. Decaluwe V, Braet C, Fairburn CG. 2003. Binge eating in obese children and adolescents. *The International journal of eating disorders* 33:78-84
44. Dekker JM, Crow RS, Folsom AR, Hannan PJ, Liao D, et al. 2000. Low heart rate variability in a 2-minute rhythm strip predicts risk of coronary heart disease and

- mortality from several causes: the ARIC Study. Atherosclerosis Risk In Communities. *Circulation* 102:1239-44
45. DiFilippo JM, Overholser JC. 2000. Suicidal ideation in adolescent psychiatric inpatients as associated with depression and attachment relationships. *J Clin Child Psychol* 29:155-66
  46. DiMatteo MR. 2004. Social support and patient adherence to medical treatment: a meta-analysis. *Health Psychol* 23:207-18
  47. Doane LD, Adam EK. 2010. Loneliness and cortisol: momentary, day-to-day, and trait associations. *Psychoneuroendocrinology* 35:430-41
  48. Doussard-Roosevelt JA, Montgomery LA, Porges SW. 2003. Short-term stability of physiological measures in kindergarten children: respiratory sinus arrhythmia, heart period, and cortisol. *Dev Psychobiol* 43:230-42
  49. Dunton GF, Liao Y, Intille SS, Spruijt-Metz D, Pentz M. 2011. Investigating children's physical activity and sedentary behavior using ecological momentary assessment with mobile phones. *Obesity (Silver Spring)* 19:1205-12
  50. Eddy KT, Tanofsky-Kraff M, Thompson-Brenner H, Herzog DB, Brown TA, Ludwig DS. 2007. Eating disorder pathology among overweight treatment-seeking youth: clinical correlates and cross-sectional risk modeling. *Behav Res Ther* 45:2360-71
  51. Egizio VB, Jennings JR, Christie IC, Sheu LK, Matthews KA, Gianaros PJ. 2008. Cardiac vagal activity during psychological stress varies with social functioning in older women. *Psychophysiology* 45:1046-54

52. Eisenberger NI, Lieberman MD. 2004. Why rejection hurts: a common neural alarm system for physical and social pain. *Trends Cogn Sci* 8:294-300
53. Eldredge KL, Agras WS. 1996. Weight and shape overconcern and emotional eating in binge eating disorder. *Int J Eat Disord* 19:73-82
54. Elliott CA, Tanofsky-Kraff M, Shomaker LB, Columbo KM, Wolkoff LE, et al. 2010. An examination of the interpersonal model of loss of control eating in children and adolescents. *Behav Res Ther* 48:424-8
55. Ellis DA, Podolski CL, Frey M, Naar-King S, Wang B, Moltz K. 2007. The role of parental monitoring in adolescent health outcomes: impact on regimen adherence in youth with type 1 diabetes. *J Pediatr Psychol* 32:907-17
56. Engel SG, Kahler KA, Lystad CM, Crosby RD, Simonich HK, Wonderlich SA. 2007. Affective patterns in eating behavior in obese BED, obese non-BED, and non-obese control participants: A naturalistic study paper. In *Eating Disorders Research Society*
57. Engel SG, Kahler KA, Lystad CM, Crosby RD, Simonich HK, et al. 2009. Eating behavior in obese BED, obese non-BED, and non-obese control participants: a naturalistic study. *Behav Res Ther* 47:897-900
58. Engel SG, Kahler KA, Lystad CM, Crosby RD, Simonich HK, et al. October, 2007. Affective patterns in eating behavior in obese BED, obese non-BED, and non-obese control participants: A naturalistic study In *Eating Disorders Research Society*. Pittsburgh

59. Engel SG, Wonderlich SA, Crosby RD, Wright TL, Mitchell JE, et al. 2005. A study of patients with anorexia nervosa using ecologic momentary assessment. *Int J Eat Disord* 38:335-9
60. Epel E, Lapidus R, McEwen B, Brownell K. 2001. Stress may add bite to appetite in women: a laboratory study of stress-induced cortisol and eating behavior. *Psychoneuroendocrinology* 26:37-49
61. Fairburn CG, Cooper Z, eds. 1993. *The Eating Disorder Examination (12th ed.)*. New York: Guilford Press. 317–60 pp. 12 ed.
62. Fairburn CG, Cooper Z, Doll HA, Norman P, O'Connor M. 2000. The natural course of bulimia nervosa and binge eating disorder in young women. *Arch Gen Psychiatry* 57:659-65
63. Fallon EM, Tanofsky-Kraff M, Norman AC, McDuffie JR, Taylor ED, et al. 2005. Health-related quality of life in overweight and nonoverweight black and white adolescents. *Journal of Pediatrics* 147:443-50
64. Fichter MM, Quadflieg N, Hedlund S. 2008. Long-term course of binge eating disorder and bulimia nervosa: relevance for nosology and diagnostic criteria. *Int J Eat Disord* 41:577-86
65. Field AE, Austin SB, Taylor CB, Malspeis S, Rosner B, et al. 2003. Relation between dieting and weight change among preadolescents and adolescents. *Pediatrics* 112:900-6
66. Flegal KM, Carroll MD, Ogden CL, Curtin LR. 2010. Prevalence and trends in obesity among US adults, 1999-2008. *JAMA* 303:235-41



67. Flook L, Repetti RL, Ullman JB. 2005. Classroom social experiences as predictors of academic performance. *Dev Psychol* 41:319-27
68. French SA, Story M, Neumark-Sztainer D, Downes B, Resnick M, Blum R. 1997. Ethnic differences in psychosocial and health behavior correlates of dieting, purging, and binge eating in a population-based sample of adolescent females. *Int J Eat Disord* 22:315-22
69. Friederich HC, Schild S, Schellberg D, Quenter A, Bode C, et al. 2006. Cardiac parasympathetic regulation in obese women with binge eating disorder. *Int J Obes (Lond)* 30:534-42
70. Friesen CA, Lin Z, Schurman JV, Andre L, Mc Callum RW. 2007. Autonomic nervous system response to a solid meal and water loading in healthy children: its relation to gastric myoelectrical activity. *Neurogastroenterol Motil* 19:376-82
71. Furman W, Buhrmester D. 1992. Age and sex differences in perceptions of networks of personal relationships. *Child Dev* 63:103-15
72. Gentzler AL, Santucci AK, Kovacs M, Fox NA. 2009. Respiratory sinus arrhythmia reactivity predicts emotion regulation and depressive symptoms in at-risk and control children. *Biol Psychol* 82:156-63
73. Glasofer DR, Tanofsky-Kraff M, Eddy KT, Yanovski SZ, Theim KR, et al. 2007. Binge eating in overweight treatment-seeking adolescents. *J Pediatr Psychol* 32:95-105
74. Gluck ME, Geliebter A, Hung J, Yahav E. 2004. Cortisol, hunger, and desire to binge eat following a cold stress test in obese women with binge eating disorder. *Psychosom Med* 66:876-81

75. Goldschmidt AB, Aspen VP, Sinton MM, Tanofsky-Kraff M, Wilfley DE. 2008. Disordered eating attitudes and behaviors in overweight youth. *Obesity (Silver Spring)* 16:257-64
76. Goldschmidt AB, Engel SG, Wonderlich SA, Crosby RD, Peterson CB, et al. 2012. Momentary affect surrounding loss of control and overeating in obese adults with and without binge eating disorder. *Obesity (Silver Spring)* 20:1206-11
77. Goldschmidt AB, Jones M, Manwaring JL, Luce KH, Osborne MI, et al. 2008. The clinical significance of loss of control over eating in overweight adolescents. *Int J Eat Disord* 41:153-8
78. Goossens L, Braet C, Decaluwe V. 2007. Loss of control over eating in obese youngsters. *Behav Res Ther* 45:1-9
79. Goossens L, Soenens B, Braet C. 2009. Prevalence and characteristics of binge eating in an adolescent community sample. *J Clin Child Adolesc Psychol* 38:342-53
80. Greeno CG, Wing RR, Shiffman S. 2000. Binge antecedents in obese women with and without binge eating disorder. *J Consult Clin Psychol* 68:95-102
81. Grenard JL, Stacy AW, Shiffman S, Baraldi AN, Mackinnon DP, et al. 2013. Sweetened drink and snacking cues in adolescents. A study using ecological momentary assessment. *Appetite* 67C:61-73
82. Grilo CM, White MA, Masheb RM. 2009. DSM-IV psychiatric disorder comorbidity and its correlates in binge eating disorder. *Int J Eat Disord* 42:228-34

83. Grucza RA, Przybeck TR, Cloninger CR. 2007. Prevalence and correlates of binge eating disorder in a community sample. *Comprehensive psychiatry* 48:124-31
84. Gwaltney CJ, Bartolomei R, Colby SM, Kahler CW. 2008. Ecological momentary assessment of adolescent smoking cessation: a feasibility study. *Nicotine Tob Res* 10:1185-90
85. Haedt-Matt AA, Keel PK. 2011. Revisiting the affect regulation model of binge eating: a meta-analysis of studies using ecological momentary assessment. *Psychol Bull* 137:660-81
86. Harthoorn LF, Dransfield E. 2008. Periprandial changes of the sympathetic-parasympathetic balance related to perceived satiety in humans. *Eur J Appl Physiol* 102:601-8
87. Hartmann AS, Czaja J, Rief W, Hilbert A. 2012. Psychosocial risk factors of loss of control eating in primary school children: a retrospective case-control study. *Int J Eat Disord* 45:751-8
88. Hawkins RC, Clement PF. 1984. Binge eating: Measurement problems and a conceptual model. In *The binge purge syndrome: Diagnosis, treatment, and research*, ed. RC Hawkins, WJ Fremouw, PF Clement:229-51. New York, NY: Springer. Number of 229-51 pp.
89. Hayden-Wade HA, Stein RI, Ghaderi A, Saelens BE, Zabinski MF, Wilfley DE. 2005. Prevalence, characteristics, and correlates of teasing experiences among overweight children vs. non-overweight peers. *Obes Res* 13:1381-92

90. Heatherton TF, Baumeister RF. 1991. Binge eating as escape from self-awareness. *Psychological Bulletin* 110:86-108
91. Hedeker D, Mermelstein RJ, Berbaum ML, Campbell RT. 2009. Modeling mood variation associated with smoking: an application of a heterogeneous mixed-effects model for analysis of ecological momentary assessment (EMA) data. *Addiction* 104:297-307
92. Hilbert A, Bishop ME, Stein RI, Tanofsky-Kraff M, Swenson AK, et al. 2012. Long-term efficacy of psychological treatments for binge eating disorder. *The British journal of psychiatry : the journal of mental science* 200:232-7
93. Hilbert A, Czaja J. 2009. Binge eating in primary school children: towards a definition of clinical significance. *Int J Eat Disord* 42:235-43
94. Hilbert A, Rief W, Tuschen-Caffier B, de Zwaan M, Czaja J. 2009. Loss of control eating and psychological maintenance in children: an ecological momentary assessment study. *Behav Res Ther* 47:26-33
95. Hilbert A, Tuschen-Caffier B. 2007. Maintenance of binge eating through negative mood: a naturalistic comparison of binge eating disorder and bulimia nervosa. *Int J Eat Disord* 40:521-30
96. Hilbert A, Tuschen-Caffier B, Czaja J. 2010. Eating behavior and familial interactions of children with loss of control eating: a laboratory test meal study. *Am J Clin Nutr* 91:510-8
97. Houtveen JH, Groot PF, Geus EJ. 2005. Effects of variation in posture and respiration on RSA and pre-ejection period. *Psychophysiology* 42:713-9

98. Hsu LK, Mulliken B, McDonagh B, Krupa Das S, Rand W, et al. 2002. Binge eating disorder in extreme obesity. *Int J Obes Relat Metab Disord* 26:1398-403
99. Hudson JI, Hiripi E, Pope HG, Jr., Kessler RC. 2007. The prevalence and correlates of eating disorders in the National Comorbidity Survey Replication. *Biol Psychiatry* 61:348-58
100. Hughes JW, Stoney CM. 2000. Depressed mood is related to high-frequency heart rate variability during stressors. *Psychosom Med* 62:796-803
101. IBM Corp. 2011. IBM SPSS Statistics for Windows. Armonk, NY: IBM Corp
102. Isnard P, Michel G, Frelut ML, Vila G, Falissard B, et al. 2003. Binge eating and psychopathology in severely obese adolescents. *The International journal of eating disorders* 34:235-43
103. Jacobs-Pilipski MJ, Wilfley DE, Crow SJ, Walsh BT, Lilenfeld LR, et al. 2007. Placebo response in binge eating disorder. *Int J Eat Disord* 40:204-11
104. Johnson WG, Rohan KJ, Kirk AA. 2002. Prevalence and correlates of binge eating in white and African American adolescents. *Eating Behaviors* 3:179-89
105. Judd CM, Kenny DA. 1981. Process analysis: Estimating mediation in treatment evaluations. *Evaluation Review* 5:602-19
106. Kalarchian MA, Marcus MD, Wilson GT, Labouvie EW, Brolin RE, LaMarca LB. 2002. Binge eating among gastric bypass patients at long-term follow-up. *Obesity Surgery* 12:270-5
107. Kaufman J, Birmaher B, Brent D, Rao U, Flynn C, et al. 1997. Schedule for Affective Disorders and Schizophrenia for School-Age Children-Present and

- Lifetime Version (K-SADS-PL): initial reliability and validity data. *J Am Acad Child Adolesc Psychiatry* 36:980-8
108. Kemp AH, Quintana DS, Gray MA, Felmingham KL, Brown K, Gatt JM. 2010. Impact of depression and antidepressant treatment on heart rate variability: a review and meta-analysis. *Biol Psychiatry* 67:1067-74
109. Kenardy J, Arnow B, Agras WS. 1996. The aversiveness of specific emotional states associated with binge-eating in obese subjects. *Aust N Z J Psychiatry* 30:839-44
110. Kennedy SH, Heslegrave RJ. 1989. Cardiac regulation in bulimia nervosa. *J Psychiatr Res* 23:267-73
111. Kessler RC, Berglund PA, Chiu WT, Deitz AC, Hudson JI, et al. 2013. The prevalence and correlates of binge eating disorder in the world health organization world mental health surveys. *Biol Psychiatry* 73:904-14
112. Kistner J, Balthazor M, Risi S, Burton C. 1999. Predicting dysphoria in adolescence from actual and perceived peer acceptance in childhood. *J Clin Child Psychol* 28:94-104
113. Klerman GL, Weissman MM, Rounsaville BJ, Chevron E. 1984. *Interpersonal psychotherapy for depression*. New York: Basic Books
114. Kop WJ, Stein PK, Tracy RP, Barzilay JI, Schulz R, Gottdiener JS. 2010. Autonomic nervous system dysfunction and inflammation contribute to the increased cardiovascular mortality risk associated with depression. *Psychosom Med* 72:626-35



115. Kop WJ, Synowski SJ, Newell ME, Schmidt LA, Waldstein SR, Fox NA. 2010. Autonomic nervous system reactivity to positive and negative mood induction: The role of acute psychological responses and frontal electrocortical activity. *Biol Psychol*
116. Kop WJ, Verdino RJ, Gottdiener JS, O'Leary ST, Bairey Merz CN, Krantz DS. 2001. Changes in heart rate and heart rate variability before ambulatory ischemic events(1). *J Am Coll Cardiol* 38:742-9
117. Krefetz DG, Steer RA, Kumar G. 2003. Lack of age differences in the Beck Depression Inventory-II scores of clinically depressed adolescent outpatients. *Psychological reports* 92:489-97
118. Kreibig SD. 2010. Autonomic nervous system activity in emotion: A review. *Biol Psychol*
119. Kreipe RE, Goldstein B, DeKing DE, Tipton R, Kempinski MH. 1994. Heart rate power spectrum analysis of autonomic dysfunction in adolescents with anorexia nervosa. *Int J Eat Disord* 16:159-65
120. Kuczmarski RJ, Ogden CL, Guo SS, Grummer-Strawn LM, Flegal KM, et al. 2002. 2000 CDC Growth Charts for the United States: methods and development. *Vital Health Stat* 11:1-190
121. Kupersmidt JB, Coie JD. 1990. Preadolescent peer status, aggression, and school adjustment as predictors of externalizing problems in adolescence. *Child Dev* 61:1350-62
122. La Rovere MT, Bigger JT, Jr., Marcus FI, Mortara A, Schwartz PJ. 1998. Baroreflex sensitivity and heart-rate variability in prediction of total cardiac

- mortality after myocardial infarction. ATRAMI (Autonomic Tone and Reflexes After Myocardial Infarction) Investigators. *Lancet* 351:478-84
123. Lachish M, Stein D, Kaplan Z, Matar M, Faigin M, et al. 2009. Irreversibility of cardiac autonomic dysfunction in female adolescents diagnosed with anorexia nervosa after short- and long-term weight gain. *World J Biol Psychiatry* 10:503-11
  124. Laederach-Hofmann K, Mussgay L, Ruddle H. 2000. Autonomic cardiovascular regulation in obesity. *J Endocrinol* 164:59-66
  125. Laurent J, Catanzaro SJ, Joiner TE, Rudolph KD, Potter KI, et al. 1999. A measure of positive and negative affect for children: Scale development and preliminary validation. *Psychological Assessment* 11:326-38
  126. Ledoux S, Choquet M, Manfredi R. 1993. Associated factors for self-reported binge eating among male and female adolescents. *J Adolesc* 16:75-91
  127. Lewandowski L, Rieger B, Smyth J, Perry L, Gathje R. 2009. Measuring post-concussion symptoms in adolescents: feasibility of ecological momentary assessment. *Archives of clinical neuropsychology : the official journal of the National Academy of Neuropsychologists* 24:791-6
  128. Lopez C, Dubois DL. 2005. Peer victimization and rejection: investigation of an integrative model of effects on emotional, behavioral, and academic adjustment in early adolescence. *J Clin Child Adolesc Psychol* 34:25-36
  129. Lu CL, Zou X, Orr WC, Chen JD. 1999. Postprandial changes of sympathovagal balance measured by heart rate variability. *Dig Dis Sci* 44:857-61

130. Macdonald G, Leary MR. 2005. Why does social exclusion hurt? The relationship between social and physical pain. *Psychol Bull* 131:202-23
131. Mallinckrodt B, Abraham WT, Wei M, Russell DW. 2006. Advances in Testing the Statistical Significance of Mediation Effects. *Journal of Counseling Psychology* 53:372-8
132. Masheb RM, Grilo CM. 2006. Emotional overeating and its associations with eating disorder psychopathology among overweight patients with binge eating disorder. *Int J Eat Disord* 39:141-6
133. Maunder RG, Lancee WJ, Nolan RP, Hunter JJ, Tannenbaum DW. 2006. The relationship of attachment insecurity to subjective stress and autonomic function during standardized acute stress in healthy adults. *J Psychosom Res* 60:283-90
134. McCraty R, Atkinson M, Tiller WA, Rein G, Watkins AD. 1995. The effects of emotions on short-term power spectrum analysis of heart rate variability. *Am J Cardiol* 76:1089-93
135. McElhaney KB, Porter MR, Thompson LW, Allen JP. 2008. Apples and Oranges: Divergent Meanings of Parents' and Adolescents' Perceptions of Parental Influence. *J Early Adolesc* 28:206-29
136. Messerli-Burgy N, Engesser C, Lemmenmeier E, Steptoe A, Laederach-Hofmann K. 2010. Cardiovascular stress reactivity and recovery in bulimia nervosa and binge eating disorder. *Int J Psychophysiol* 78:163-8
137. Miller PJ, Niehuis S, Huston TL. 2006. Positive illusions in marital relationships: A 13-year longitudinal study. *Pers Soc Psychol Bull* 32:1579-94

138. Morgan CM, Yanovski SZ, Nguyen TT, McDuffie J, Sebring NG, et al. 2002. Loss of control over eating, adiposity, and psychopathology in overweight children. *Int J Eat Disord* 31:430-41
139. Muehlenkamp JJ, Engel SG, Wadeson A, Crosby RD, Wonderlich SA, et al. 2009. Emotional states preceding and following acts of non-suicidal self-injury in bulimia nervosa patients. *Behav Res Ther* 47:83-7
140. Mufson L, Dorta KP, Moreau D, Weissman MM. 2004. *Interpersonal Psychotherapy for Depressed Adolescents*. New York, NY: The Guilford Press
141. Mufson L, Dorta KP, Wickramaratne P, Nomura Y, Olfson M, Weissman MM. 2004. A randomized effectiveness trial of interpersonal psychotherapy for depressed adolescents. *Arch Gen Psychiatry* 61:577-84
142. Mulvaney SA, Mudasiru E, Schlundt DG, Baughman CL, Fleming M, et al. 2008. Self-management in type 2 diabetes: the adolescent perspective. *The Diabetes educator* 34:674-82
143. Mulvaney SA, Rothman RL, Dietrich MS, Wallston KA, Grove E, et al. 2012. Using mobile phones to measure adolescent diabetes adherence. *Health Psychol* 31:43-50
144. Murialdo G, Casu M, Falchero M, Brugnolo A, Patrone V, et al. 2007. Alterations in the autonomic control of heart rate variability in patients with anorexia or bulimia nervosa: correlations between sympathovagal activity, clinical features, and leptin levels. *J Endocrinol Invest* 30:356-62

145. Muris P, Merckelbach H, Ollendick T, King N, Bogie N. 2002. Three traditional and three new childhood anxiety questionnaires: their reliability and validity in a normal adolescent sample. *Behav Res Ther* 40:753-72
146. Natsuaki MN, Klimes-Dougan B, Ge X, Shirtcliff EA, Hastings PD, Zahn-Waxler C. 2009. Early pubertal maturation and internalizing problems in adolescence: sex differences in the role of cortisol reactivity to interpersonal stress. *J Clin Child Adolesc Psychol* 38:513-24
147. Neumark-Sztainer D, Story M, French SA, Hannan PJ, Resnick MD, Blum RW. 1997. Psychosocial concerns and health-compromising behaviors among overweight and nonoverweight adolescents. *Obesity Research* 5:237-49
148. Neumark-Sztainer D, Story M, Hannan PJ, Perry CL, Irving LM. 2002. Weight-related concerns and behaviors among overweight and nonoverweight adolescents: implications for preventing weight-related disorders. *Arch Pediatr Adolesc Med* 156:171-8
149. Nock MK, Prinstein MJ, Sterba SK. 2009. Revealing the form and function of self-injurious thoughts and behaviors: A real-time ecological assessment study among adolescents and young adults. *J Abnorm Psychol* 118:816-27
150. Ogden CL, Carroll MD, Kit BK, Flegal KM. 2012. Prevalence of obesity and trends in body mass index among US children and adolescents, 1999-2010. *JAMA* 307:483-90
151. Ogden CL, Kuczmarski RJ, Flegal KM, Mei Z, Guo S, et al. 2002. Centers for Disease Control and Prevention 2000 growth charts for the United States:

improvements to the 1977 National Center for Health Statistics version.

*Pediatrics* 109:45-60

152. Oliver G, Wardle J, Gibson EL. 2000. Stress and food choice: a laboratory study. *Psychosom Med* 62:853-65
153. Pearce MJ, Boergers J, Prinstein MJ. 2002. Adolescent obesity, overt and relational peer victimization, and romantic relationships. *Obes Res* 10:386-93
154. Perini R, Milesi S, Biancardi L, Veicsteinas A. 1996. Effects of high altitude acclimatisation on heart rate variability in resting humans. *Eur J Appl Physiol* 73:521-8
155. Perini R, Veicsteinas A. 2003. Heart rate variability and autonomic activity at rest and during exercise in various physiological conditions. *European journal of applied physiology* 90:317-25
156. Pieters G, Vansteelandt K, Claes L, Probst M, Van Mechelen I, Vandereycken W. 2006. The usefulness of experience sampling in understanding the urge to move in anorexia nervosa. *Acta Neuropsychiatrica* 18:30-7
157. Pinaquy S, Chabrol H, Simon C, Louvet JP, Barbe P. 2003. Emotional eating, alexithymia, and binge-eating disorder in obese women. *Obes Res* 11:195-201
158. Porges SW. 1995. Cardiac vagal tone: a physiological index of stress. *Neurosci Biobehav Rev* 19:225-33
159. Prinstein MJ, Boergers J, Spirito A, Little TD, Grapentine WL. 2000. Peer functioning, family dysfunction, and psychological symptoms in a risk factor model for adolescent inpatients' suicidal ideation severity. *J Clin Child Psychol* 29:392-405



160. Radhakrishna RK, Dutt DN, Yeragani VK. 2000. Nonlinear measures of heart rate time series: influence of posture and controlled breathing. *Auton Neurosci* 83:148-58
161. Ranzenhofer LM, Hannallah L, Field SE, Shomaker LB, Stephens M, et al. 2013. Pre-meal affective state and laboratory test meal intake in adolescent girls with loss of control eating. *Appetite* 68:30-7
162. Ranzenhofer LM, Tanofsky-Kraff M, McDuffie J, Salaita CG, Yanoff LB, et al. unpublished work. Title. Volume:In press
163. Rechlin T, Weis M, Ott C, Bleichner F, Joraschky P. 1998. Alterations of autonomic cardiac control in anorexia nervosa. *Biol Psychiatry* 43:358-63
164. Rechlin T, Weis M, Spitzer A, Kaschka WP. 1994. Are affective disorders associated with alterations of heart rate variability? *J Affect Disord* 32:271-5
165. Ricca V, Castellini G, Lo Sauro C, Ravaldi C, Lapi F, et al. 2009. Correlations between binge eating and emotional eating in a sample of overweight subjects. *Appetite* 53:418-21
166. Rieger E, Wilfley DE, Stein RI, Marino V, Crow SJ. 2005. A comparison of quality of life in obese individuals with and without binge eating disorder. *Int J Eat Disord* 37:234-40
167. Rissanen A, Naukkarinen H, Virkkunen M, Rawlings RR, Linnoila M. 1998. Fluoxetine normalizes increased cardiac vagal tone in bulimia nervosa. *J Clin Psychopharmacol* 18:26-32
168. Rizvi SL, Peterson CB, Crow SJ, Agras WS. 2000. Test-retest reliability of the eating disorder examination. *Int J Eat Disord* 28:311-6

169. Rossello J, Bernal G. 1999. The efficacy of cognitive-behavioral and interpersonal treatments for depression in Puerto Rican adolescents. *J Consult Clin Psychol* 67:734-45
170. Rudolph KD. 2002. Gender differences in emotional responses to interpersonal stress during adolescence. *J Adolesc Health* 30:3-13
171. Rueter MA, Scaramella L, Wallace LE, Conger RD. 1999. First onset of depressive or anxiety disorders predicted by the longitudinal course of internalizing symptoms and parent-adolescent disagreements. *Arch Gen Psychiatry* 56:726-32
172. Rusby JC, Westling E, Crowley R, Light JM. 2013. Concurrent and predictive associations between early adolescent perceptions of peer affiliates and mood states collected in real time via ecological momentary assessment methodology. *Psychol Assess* 25:47-60
173. Ryan AD, Larsen PD, Galletly DC. 2003. Comparison of heart rate variability in supine, and left and right lateral positions. *Anaesthesia* 58:432-6
174. Scagliusi FB, Ferrioli E, Pfrimer K, Laureano C, Cunha CS, et al. 2009. Characteristics of women who frequently under report their energy intake: a doubly labelled water study. *European journal of clinical nutrition* 63:1192-9
175. Schmitz J, Tuschen-Caffier B, Wilhelm FH, Blechert J. 2013. Taking a closer look: autonomic dysregulation in socially anxious children. *European child & adolescent psychiatry*
176. Schwartz JE, Stone AA. 1998. Strategies for analyzing ecological momentary assessment data. *Health Psychol* 17:6-16

177. Schwerdtfeger A, Friedrich-Mai P. 2009. Social interaction moderates the relationship between depressive mood and heart rate variability: evidence from an ambulatory monitoring study. *Health Psychol* 28:501-9
178. Schwimmer JB, Burwinkle TM, Varni JW. 2003. Health-related quality of life of severely obese children and adolescents. *JAMA* 289:1813-9
179. Shiffman S, Paty JA, Gnys M, Kassel JA, Hickcox M. 1996. First lapses to smoking: Within-subjects analysis of real-time reports. *J Consult Clin Psychol* 64:366-79
180. Shiffman S, Stone AA, Hufford MR. 2008. Ecological momentary assessment. *Annu Rev Clin Psychol* 4:1-32
181. Shomaker LB, Tanofsky-Kraff M, Elliott C, Wolkoff LE, Columbo KM, et al. 2009. Salience of loss of control for pediatric binge episodes: Does size really matter? *Int J Eat Disord*
182. Shrout PE, Bolger N. 2002. Mediation in experimental and nonexperimental studies: new procedures and recommendations. *Psychol Methods* 7:422-45
183. Siebert J, Drabik P, Lango R, Szyndler K. 2004. Stroke volume variability and heart rate power spectrum in relation to posture changes in healthy subjects. *Med Sci Monit* 10:MT31-7
184. Silk JS, Forbes EE, Whalen DJ, Jakubcak JL, Thompson WK, et al. 2011. Daily emotional dynamics in depressed youth: a cell phone ecological momentary assessment study. *Journal of experimental child psychology* 110:241-57

185. Smith DE, Marcus MD, Lewis CE, Fitzgibbon M, Schreiner P. 1998. Prevalence of binge eating disorder, obesity, and depression in a biracial cohort of young adults. *Ann Behav Med* 20:227-32
186. Smyth J, Ockenfels MC, Porter L, Kirschbaum C, Hellhammer DH, Stone AA. 1998. Stressors and mood measured on a momentary basis are associated with salivary cortisol secretion. *Psychoneuroendocrinology* 23:353-70
187. Smyth J, Wonderlich S, Heron KE, Sliwinski MJ, Crosby R, et al. 2008. Ecological momentary assessment of affect, stress, and binge-purge behaviors: Day of week and time of day effects in the natural environment. *Int J Eat Disord* 42:429 - 36
188. Smyth J, Wonderlich SA, Heron KE, Sliwinski MJ, Crosby RD, et al. 2007. Daily and momentary mood and stress are associated with binge eating and vomiting in bulimia nervosa patients in the natural environment. *J Consult Clin Psychol* 75:629-38
189. Sobel ME. 1982. Asymptotic confidence interval for indirect effects in structural equation models. In *Sociological methodology*, ed. S Leinhardt:290-312. San Francisco: Jossey-Bass. Number of 290-312 pp.
190. Spielberger CD, Edwards CD, Lushene RE, Montuori J, Platzek D. 1973. *STIAC preliminary manual*. Palo Alto, CA: Consulting Psychologists Press
191. Spitzer RL, Devlin M, Walsh TB, Hasin D, Wing R, et al. 1992. Binge eating disorder: a multisite field trial of the diagnostic criteria. *Int J Eat Disord* 11:191-203

192. Spitzer RL, Yanovski S, Wadden T, Wing R, Marcus MD, et al. 1993. Binge eating disorder: its further validation in a multisite study. *Int J Eat Disord* 13:137-53
193. Steiger H, Puentes-Neuman G, Leung FY. 1991. Personality and family features of adolescent girls with eating symptoms: evidence for restrictor/binger differences in a nonclinical population. *Addict Behav* 16:303-14
194. Stein RI, Kenardy J, Wiseman CV, Douchis JZ, Arnow BA, Wilfley DE. 2007. What's driving the binge in binge eating disorder?: A prospective examination of precursors and consequences. *Int J Eat Disord* 40:195-203
195. Steinberg L. 2005. Cognitive and affective development in adolescence. *Trends Cogn Sci* 9:69-74
196. Stice E, Cameron RP, Killen JD, Hayward C, Taylor CB. 1999. Naturalistic weight-reduction efforts prospectively predict growth in relative weight and onset of obesity among female adolescents. *J Consult Clin Psychol* 67:967-74
197. Stone AA, Broderick JE, Porter LS, Kaell AT. 1997. The experience of rheumatoid arthritis pain and fatigue: Examining momentary reports and correlates over one week. *Arthritis Care Res* 10:185-93
198. Strauss RS, Pollack HA. 2003. Social marginalization of overweight children. *Arch Pediatr Adolesc Med* 157:746-52
199. Striegel-Moore RH, Wilfley DE, Pike KM, Dohm FA, Fairburn CG. 2000. Recurrent binge eating in black American women. *Arch Fam Med* 9:83-7
200. Stroud LR, Salovey P, Epel ES. 2002. Sex differences in stress responses: social rejection versus achievement stress. *Biol Psychiatry* 52:318-27

201. Stroud LR, Tanofsky-Kraff M, Wilfley DE, Salovey P. 2000. The Yale Interpersonal Stressor (YIPS): affective, physiological, and behavioral responses to a novel interpersonal rejection paradigm. *Ann Behav Med* 22:204-13
202. Sullivan HS. 1953. *The Interpersonal Theory of Psychiatry*. New York: Norton
203. Tamir M, Robinson MD. 2004. Knowing good from bad: the paradox of neuroticism, negative affect, and evaluative processing. *J Pers Soc Psychol* 87:913-25
204. Tanofsky-Kraff M. 2008. Binge eating among children and adolescents. In *Handbook of Child and Adolescent Obesity*, ed. E Jelalian, R Steele:41-57; New York: Springer Publishers. Number of 41-57 pp.
205. Tanofsky-Kraff M, Cohen ML, Yanovski SZ, Cox C, Theim KR, et al. 2006. A prospective study of psychological predictors of body fat gain among children at high risk for adult obesity. *Pediatrics* 117:1203-9
206. Tanofsky-Kraff M, Faden D, Yanovski SZ, Wilfley DE, Yanovski JA. 2005. The perceived onset of dieting and loss of control eating behaviors in overweight children. *Int J Eat Disord* 38:112-22
207. Tanofsky-Kraff M, Goossens L, Eddy KT, Ringham R, Goldschmidt A, et al. 2007. A multisite investigation of binge eating behaviors in children and adolescents. *Journal of consulting and clinical psychology* 75:901-13
208. Tanofsky-Kraff M, McDuffie JR, Yanovski SZ, Kozlosky M, Schvey NA, et al. 2009. Laboratory assessment of the food intake of children and adolescents with loss of control eating. *Am J Clin Nutr* 89:738-45



209. Tanofsky-Kraff M, Morgan CM, Yanovski SZ, Marmarosh C, Wilfley DE, Yanovski JA. 2003. Comparison of assessments of children's eating-disordered behaviors by interview and questionnaire. *The International journal of eating disorders* 33:213-24
210. Tanofsky-Kraff M, Shomaker LB, Roza CA, Wolkoff LE, Columbo KM, et al. 2011. A Prospective Study of Pediatric Loss of Control Eating and Psychological Outcomes. *Journal of Abnormal Psychology*
211. Tanofsky-Kraff M, Theim KR, Yanovski SZ, Bassett AM, Burns NP, et al. 2007. Validation of the emotional eating scale adapted for use in children and adolescents (EES-C). *Int J Eat Disord* 40:232-40
212. Tanofsky-Kraff M, Wilfley DE, Young JF, Mufson L, Yanovski SZ, et al. 2007. Preventing excessive weight gain in adolescents: interpersonal psychotherapy for binge eating. *Obesity (Silver Spring)* 15:1345-55
213. Tanofsky-Kraff M, Wilfley DE, Young JF, Mufson L, Yanovski SZ, et al. 2009. A pilot study of interpersonal psychotherapy for preventing excess weight gain in adolescent girls at-risk for obesity. *Int J Eat Disord*
214. Tanofsky-Kraff M, Yanovski SZ, Schvey NA, Olsen CH, Gustafson J, Yanovski JA. 2009. A prospective study of loss of control eating for body weight gain in children at high risk for adult obesity. *Int J Eat Disord* 42:26-30
215. Tanofsky-Kraff M, Yanovski SZ, Wilfley DE, Marmarosh C, Morgan CM, Yanovski JA. 2004. Eating-disordered behaviors, body fat, and psychopathology in overweight and normal-weight children. *Journal of consulting and clinical psychology* 72:53-61

216. Task Force of the European Society of Cardiology and the North American Society of Pacing and Electrophysiology. 1996. Heart Rate Variability: Standards of Measurement, Physiological Interpretation, and Clinical Use. *Circulation* 93:1043-65
217. Tataranni PA, Larson DE, Snitker S, Young JB, Flatt JP, Ravussin E. 1996. Effects of glucocorticoids on energy metabolism and food intake in humans. *Am J Physiol* 271:E317-25
218. Taylor GJ, Bagby RM, Parker JDA. 1997. *Disorders of affect regulation: alexithymia in medical and psychiatric illness*. Cambridge: Cambridge University Press
219. Telch CF, Agras WS. 1996. Do emotional states influence binge eating in the obese? *Int J Eat Disord* 20:271-9
220. Telch CF, Stice E. 1998. Psychiatric comorbidity in women with binge eating disorder: prevalence rates from a non-treatment-seeking sample. *J Consult Clin Psychol* 66:768-76
221. Thayer JF, Lane RD. 2000. A model of neurovisceral integration in emotion regulation and dysregulation. *J Affect Disord* 61:201-16
222. The Rudd Center for Food Policy and Obesity. 2012. Marketing Unhealthy Food and Beverages to Youth via Mobile Devices. ed. Y University
223. Tonhajzerova I, Ondrejka I, Javorka K, Turianikova Z, Farsky I, Javorka M. 2010. Cardiac autonomic regulation is impaired in girls with major depression. *Prog Neuropsychopharmacol Biol Psychiatry* 34:613-8

224. Tonhajzerova I, Ondrejka I, Javorka M, Adamik P, Turianikova Z, et al. 2009. Respiratory sinus arrhythmia is reduced in adolescent major depressive disorder. *Eur J Med Res* 14 Suppl 4:280-3
225. Tsuji H, Larson MG, Venditti FJ, Jr., Manders ES, Evans JC, et al. 1996. Impact of reduced heart rate variability on risk for cardiac events. The Framingham Heart Study. *Circulation* 94:2850-5
226. Turner SA, Luszczynska A, Warner L, Schwarzer R. 2010. Emotional and uncontrolled eating styles and chocolate chip cookie consumption. A controlled trial of the effects of positive mood enhancement. *Appetite* 54:143-9
227. Valensi P, Thi BN, Lormeau B, Paries J, Attali JR. 1995. Cardiac autonomic function in obese patients. *Int J Obes Relat Metab Disord* 19:113-8
228. Van Strien T, Engels RC, Van Leeuwe J, Snoek HM. 2005. The Stice model of overeating: tests in clinical and non-clinical samples. *Appetite* 45:205-13
229. Vogele C, Hilbert A, Tuschen-Caffier B. 2009. Dietary restriction, cardiac autonomic regulation and stress reactivity in bulimic women. *Physiol Behav* 98:229-34
230. Vogele C, Sorg S, Studtmann M, Weber H. 2010. Cardiac autonomic regulation and anger coping in adolescents. *Biol Psychol* 85:465-71
231. Wang Y, Zhao X, O'Neil A, Turner A, Liu X, Berk M. 2013. Altered cardiac autonomic nervous function in depression. *BMC psychiatry* 13:187
232. Watanabe N, Reece J, Polus BI. 2007. Effects of body position on autonomic regulation of cardiovascular function in young, healthy adults. *Chiropr Osteopat* 15:19

233. Wegner KE, Smyth JM, Crosby RD, Wittrock D, Wonderlich SA, Mitchell JE. 2002. An evaluation of the relationship between mood and binge eating in the natural environment using ecological momentary assessment. *Int J Eat Disord* 32:352-61
234. Weinstein AA, Deuster PA, Kop WJ. 2007. Heart rate variability as a predictor of negative mood symptoms induced by exercise withdrawal. *Med Sci Sports Exerc* 39:735-41
235. Weissman MM, Bothwell S. 1976. Assessment of social adjustment by patient self-report. *Arch Gen Psychiatry* 33:1111-5
236. Whalen CK, Henker B, Jamner LD, Ishikawa SS, Floro JN, et al. 2006. Toward mapping daily challenges of living with ADHD: maternal and child perspectives using electronic diaries. *J Abnorm Child Psychol* 34:115-30
237. Wilfley DE, Schwartz MB, Spurrell EB, Fairburn CG. 2000. Using the eating disorder examination to identify the specific psychopathology of binge eating disorder. *Int J Eat Disord* 27:259-69
238. Wilfley DE, Welch RR, Stein RI, Spurrell EB, Cohen LR, et al. 2002. A randomized comparison of group cognitive-behavioral therapy and group interpersonal psychotherapy for the treatment of overweight individuals with binge-eating disorder. *Arch Gen Psychiatry* 59:713-21
239. Witvliet M, Brendgen M, van Lier PA, Koot HM, Vitaro F. 2010. Early Adolescent Depressive Symptoms: Prediction from Clique Isolation, Loneliness, and Perceived Social Acceptance. *J Abnorm Child Psychol*

- 240. Wolfe BE, Baker CW, Smith AT, Kelly-Weeder S. 2009. Validity and utility of the current definition of binge eating. *Int J Eat Disord* 42:674-86
- 241. Yanovski SZ, Nelson JE, Dubbert BK, Spitzer RL. 1993. Association of binge eating disorder and psychiatric comorbidity in obese subjects. *JAMA Psychiatry* 150:1472-9
- 242. Yin J, Levanon D, Chen JD. 2004. Inhibitory effects of stress on postprandial gastric myoelectrical activity and vagal tone in healthy subjects. *Neurogastroenterol Motil* 16:737-44
- 243. Young JF, Mufson L, Davies M. 2006. Efficacy of Interpersonal Psychotherapy-Adolescent Skills Training: an indicated preventive intervention for depression. *J Child Psychol Psychiatry* 47:1254-62
- 244. Young JF, Mufson L, Gallop R. 2010. Preventing depression: a randomized trial of interpersonal psychotherapy-adolescent skills training. *Depress Anxiety* 27:426-33

# Appendix I.



## Evaluation Daily Diary Study

ID

Date:   /   /

1. How easy did you find the monitoring to be?

not at all easy ○ ○ ○ ○ ○ ○ ○ ○ extremely easy

2. How disruptive did you find the monitoring to be?

not at all disruptive ○ ○ ○ ○ ○ ○ ○ ○ extremely disruptive

3. How time consuming did you find the monitoring to be?

not at all time consuming ○ ○ ○ ○ ○ ○ ○ ○ extremely time consuming

4. How helpful has the monitoring been in helping you better understand your mood, eating behavior, and coping patterns?

not at all helpful ○ ○ ○ ○ ○ ○ ○ ○ extremely helpful

5. How would you describe your experience in self-monitoring?

very negative ○ ○ ○ ○ ○ ○ ○ ○ very positive

6. What were the most common reasons you were not able to complete ratings when you were asked to?

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Comments: 

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# Teen Girls and Parents!!!

**Do you worry about your teen's eating and gaining too much weight?**

**Be a volunteer for a research study about emotions and eating!**



- **WHAT:** To explore teens' day to day patterns in their relationships, mood, and eating habits, using palm pilots that teens carry for two weeks.
- **WHO:** Girls aged 12-17 years who are overweight and report loss of control eating.
- **WHERE:** The Uniformed Services University of the Health Sciences, Bethesda, MD, just outside of Washington, DC.

**Be compensated (\$\$) for your time**

**All visits on weekends/after school!**

**For more information please contact: Lisa Ranzenhofer**

USUHS IRB APPROVED  
15 NOVEMBER 2012  
Expires: 17 FEBRUARY 2013

**(301) 295-2853**

**[lisa.ranzenhofer@usuhs.edu](mailto:lisa.ranzenhofer@usuhs.edu)**

Appendix 3.



Uniformed Services University of the Health Sciences,  
4301 Jones Bridge Road,  
Bethesda, Maryland 20814-4712

Season, year

Dear Parent:

We are reaching out to families in Washington, D.C. and the surrounding area to let them know about a research study we are conducting. If this information is of interest to you, please contact us; otherwise, please discard this letter.

We are conducting a study to understand eating behaviors and emotions in adolescent girls. The study takes place at the Uniformed Services University of the Health Sciences, in Bethesda, Maryland. Participants will be compensated for their time.

If you think your daughter is above average weight, and might be interested in hearing more about this study, please see the enclosed flyer and call 301-295-2853. Thank you!

Sincerely,

A handwritten signature in black ink, appearing to read 'Lisa Ranzenhofer'.

Lisa Ranzenhofer, MS  
Study Contact  
301-295-2853  
lisa.ranzenhofer@usuhs.edu

A handwritten signature in black ink, appearing to read 'Marian Tanofsky-Kraff'.

Marian Tanofsky-Kraff, PhD

## Appendix 4.

### Interpersonal problems

Since you were last beeped,  
how much have you argued  
with someone?

Not at all      Some      A lot

1	2	3	4	5
---	---	---	---	---

Since you were last beeped,  
how rejected have you felt?

Not at all      Some      A lot

1	2	3	4	5
---	---	---	---	---

Prev Page

Next Page

Since you were last beeped,  
how lonely have you felt?

Not at all      Some      A lot

1	2	3	4	5
---	---	---	---	---

Since you were last beeped,  
how much have you wished  
your relationships were  
better?

Not at all      Some      A lot

1	2	3	4	5
---	---	---	---	---

Prev Page

Next Page

Since you were last beeped,  
how much have you wished  
you had more friends?

Not at all      Some      A lot

1	2	3	4	5
---	---	---	---	---

Prev Page

Next Page

## Negative and Positive Affect

Please indicate how much you feel this way right now:

	Not much or not at all	Some	A lot	
Happy	1	2	3	4 5
Upset	1	2	3	4 5
Cheerful	1	2	3	4 5
Scared	1	2	3	4 5
Proud	1	2	3	4 5

Prev Page

Next Page

Please indicate how much you feel this way right now:

	Not much or not at all	Some	A lot	
Miserable	1	2	3	4 5
Joyful	1	2	3	4 5
Lonely	1	2	3	4 5
Delighted	1	2	3	4 5
Blue	1	2	3	4 5

Prev Page

Next Page

Please indicate how much you feel this way right now:

	Not much or not at all	Some	A lot	
Lively	1	2	3	4 5
Sad	1	2	3	4 5
Boredom	1	2	3	4 5

Prev Page

Next Page

LOC eating

To what degree did you lose control during this eating episode?

Not at all      Some      A lot

1	2	3	4	5
---	---	---	---	---

Did you feeling like you eating was out of control AT ANY POINT during the meal you just finished?

Not at all      Some      A lot

1	2	3	4	5
---	---	---	---	---

Prev Page

Next Page

During the eating episode you just finished, did you feel a sense of loss of control?

Not at all      Some      Very much

1	2	3	4	5
---	---	---	---	---

Did it feel like you were able to stop eating during the meal you just finished?

Not at all      Some      Very much

1	2	3	4	5
---	---	---	---	---

Prev Page

Next Page